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ON CHRONIC ATROPHIC SPINAL PARALYSIS IN CHILDREN.

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In the number of 'BRAIN' for April 1883, English readers were gratified by an article from Professor Erb of Heidelberg, entitled "On Chronic Atrophic Spinal Paralysis in the Child." The views of so distinguished a neurologist will have been received with the attention and respect which they merit. Having had some experience in this interesting clinical condition, I venture to make a few observations on the subject, with special reference to this paper.

In the first place, I would remark that, throughout what is to follow, the expression "Atrophic Spinal Paralysis" is adopted only because it is the terminology employed by the German author, and not because I consider the name a suitable one. In my opinion it embraces too much, applying, as it does, to a somewhat extensive group of diseases, and might include any form of paralysis accompanied with muscular wasting, due to a lesion of the spinal cord, such, for example, as progressive muscular atrophy. A new and more special nomenclature is therefore required clearly to differentiate this particular state from other allied but distinct clinical conditions. In the present paper I understand by the term atrophic

spinal paralysis that disease which is more commonly called "chronic anterior poliomyelitis," "chronic infantile paralysis," or the "paralysie spinale antérieure subaiguë" of Duchenne.

The object of Professor Erb's remarks is to show that chronic anterior poliomyelitis, although by no means uncommon in the adult, is (unlike its acute prototype) almost an unknown disease in young children, and he records in detail the facts of a case which has occurred in his practice. In referring to the literature of the subject, he points out that, with the exception of an instance in a girl of fifteen years old described by Dr. Ross, there is no example of the kind hitherto published, except one referred to by myself in my work on electro-diagnosis.[1]

He, however, questions the correctness of the diagnosis in this particular instance, and considers the case which he puts forward as "unique," regarding the youth of the patient, namely six years of age. That I at the time recognised the true nature of the affection in a child two years old is evidenced by the fact that the observation is headed "poliomyelitis anterior chronica." I am willing, however, to admit that the details of the case were not given with sufficient extension to establish the diagnosis, to the satisfaction of others, as my object was not to prove the rarity of the affection, but only to instance certain physical electrical phenomena in an individual muscle of the patient in question. Further, I did not at the time fully recognise the importance of the case itself as being of such exceptional occurrence, as I believed that I had seen other examples of a similar nature. Fortunately I have retained the full notes of this particular patient; and as so experienced an observer as Professor Erb pronounces an instance of the disease to be of such great interest, I make no excuse for recording the case shortly in detail, which will, I think, establish my original diagnosis to have been correct. I shall also add the records of others which have come under my observation and which I believe to belong to the same category. The facts of the disputed case are as follows.

[1] 'A Practical Treatise on Electro-diagnosis in Diseases of the Nervous System,' p. 121; H. K. Lewis, London, 1882.

I. *Case of Chronic Atrophic Spinal Paralysis in a Child two years of age.*

Charles S., æt. 2, was brought to the Westminster Hospital on the 2nd of November, 1880. The patient was stated to have been in good health till he was eighteen months of age. When a year old he began to walk, and soon after he was able to run about like other children, and this continued till he was one and a half years old. About this time he suffered from whooping-cough and measles, from which, however, he rapidly recovered. A month afterwards it was for the first time noticed that there was slight weakness and unsteadiness in his gait, and that he had a tendency to fall down when he ran. These symptoms, scarcely noticeable at first, gradually increased in severity, and have continued to do so up to the present time. Latterly it has been observed that the patient has been awkward with his hands, but the parents have noticed no other peculiarity of the upper extremities. The general health of the child has been uniformly good, and, with the above exceptions, he has never had a day's illness in his life.

At present the appearance and general condition of the patient are healthy in every respect. He is fat and well-nourished; there is no spinal curvature; and all his functions, except those to be described, are normal. When placed on his legs, he can barely support the weight of the body, even with assistance. The limbs are weak and shaky; attempts to walk are very uncertain and imperfect, and only clumsily effected when the patient is partially supported. When sitting on his mother's knee, the lower limbs are moved about, but not with the vigour of health. The movements of the arms are active, but the actions of the hands and fingers are clumsy, and the child picks up a penny from the table in an awkward manner. Owing to his youth, it is difficult to determine exactly the precise deficiency of these movements. For the same reason the sensibility of the skin cannot be accurately ascertained; but, so far as can be made out by touching the child unawares, or pricking him when he is not looking, and otherwise testing its condition, it seems normal, as his attention is attracted by the slightest touch, and he is irritated by the

gentlest prick or pinch. The plantar reflexes are well developed; the cremaster phenomenon distinctly marked; and the knee-jerk present on both sides, but not exaggerated. The limbs and joints are flaccid, and there is no trace of rigidity. The appearance of the skin of the upper limbs is normal. Here, as well as all over the body, there is a great development of fat; but, in spite of this, distinct atrophy of the extensors of the forearms and the small muscles of the hands on both sides is visible. There is no apparent loss of bulk in the muscles of the lower extremities, but the skin, especially on the left side, is cold and cyanotic. There is no affection of the bladder or rectum, nor are there any cerebral symptoms.

Electrical reactions.—As far as can be determined in a restless fretful child, they are as follows. The *faradic* current, when applied to the trunks of the ulnar nerves, causes on both sides normal muscular contractions. The reactions to both median nerves are considerably diminished, but not lost to a strong current. The greatest power of the battery fails to produce any effect on either musculo-spiral nerve, when irritated in the upper arm above the elbow-joint. A moderate current applied directly to the flexors of the forearms causes apparently healthy contractions; but, even when very strong, to the extensors it fails to induce any result except slight action of the extensor indicis on the right, and feeble contraction of the extensor ulnaris on the left side. To the other extensors the most powerful currents are followed by negative results. The *galvanic* current induces apparently normal contractions to the trunks of both ulnar and median nerves. A very strong current to the musculo-spiral nerves causes extension of the wrist and fingers, but slowly and imperfectly, KCC > ACC, but the latter increased. The galvanic current applied directly to the flexors of the forearms induces vigorous contractions, and the ACC is distinctly increased. To the extensor indicis of the right side the response is diminished, ACC > KCC, the contraction being slow and tonic. The same is observed in the extensor ulnaris of the left side. To the other extensors of the wrist and fingers and the small muscles of the hand, the strongest galvanic current fails to induce any contractions. Electrical investigation of the nerve-trunks and muscles of the

lower limbs proves them to be normal, as also are those of the trunk and upper arms. Galvanometric observations show that the difference in the muscular contractions is not due to any special abnormal resistance.

May 1st, 1883.—The patient was seen on this date, that is, nearly two years and a half after the foregoing report was made. He was found in perfect health and without a trace of paralysis, or other abnormal symptoms. The mother states that for six months he was kept in bed and treated with galvanism to the spine, that improvement gradually ensued, and that at the end of this time he had completely recovered.

Although the record of the facts of this case is admittedly not perfect, as it is almost impossible to conduct delicate sensory and electrical investigations in a child only two years of age, they appear to me to furnish sufficient evidence as to the nature of the lesion, namely a chronic anterior poliomyelitis, probably limited to the lower cervical and upper dorsal regions of the cord. The disease began insidiously, and progressed slowly; it was attended with weakness and wasting of the upper extremities, with no apparent illness of any kind, with no alteration in the general health or nutrition, and with no abnormal sensory symptoms or disturbance of the bladder or rectum. It was accompanied with positive electrical abnormalities in the nerve-trunks and muscles of the upper limbs, as already described. The lower extremities, although paretic, were not wasted; they possessed their sensibility and reflex actions intact, and the electrical reactions were normal. The weakness of the legs was therefore probably not the result of disease of the grey matter of the corresponding segments of the cord, but due to interruption of the voluntary impulses by the disease at a higher level. This is contrary to the usual ascending nature of the disease; but instances are not wanting in the adult, and referred to more especially by Duchenne, where the affection was descending in nature, beginning in the upper and spreading to the lower extremities. The exact limits of the poliomyelitis could not, under the circumstances, be fully determined, owing to the difficult and uncertain task of investigating so young a subject. Although there was no actual confir-

mation of the diagnosis in this case, the patient having recovered, the above seems the most plausible explanation of the facts, namely that it is an example of limited chronic atrophic spinal paralysis.

The following is also a case which has recently come under my observation.

II. *Case of Chronic Atrophic Spinal Paralysis in a Child one and a half years old.*

A. C., æt. 1½ years, male, was seen by me, in consultation with Dr. Bristowe, on the 11th of October, 1882. The patient is the child of a medical man, from whom a very accurate and trustworthy account was obtained. From his birth he has been in uniformly good health, and nothing in his condition attracted attention till he was about eight or ten months old. It was then observed that although he moved his limbs freely, they were weak, and the legs did not in any way serve to support him when he was placed upright. The entire body, moreover, was flaccid and limp, so that he could not maintain a sitting posture, and he fell about "all of a heap." This condition has slowly and gradually increased till the present time, and the child has never been able to walk, or even stand or sit upright. There has been no fever nor illness of any kind, no pain nor other abnormal symptoms, and the functions and general nutrition of the body have been uninterruptedly good.

At present, the patient is a plump, rosy, healthy-looking child. The intelligence, as far as can be ascertained, is normal. The movements of the upper extremities seem to be naturally performed, but it is very difficult to estimate any delicate changes in so young a child. The muscles of the neck and trunk are evidently weak. The head has a tendency to fall in various directions, and the patient prefers to keep it supported by his nurse's arm. The child cannot sit up by himself, but falls down "all of a heap," generally forwards, and when placed on his face or back, he cannot raise himself into an upright posture. The lower limbs are voluntarily moved about, but not with the vigour of health, and the patient

cannot support the weight of his body on his legs, which sink beneath the effort.

The plantar reflexes are totally absent, and there is no trace of the knee-jerk phenomenon on either side. There is no curvature of the spine, but the back is much rounded, evidently from the habit which the child has of lying in a forward position. As far as can be determined, the sensibility is not impaired. There are no definite vaso-motor changes, nor is there any visible wasting of the limbs; but it must be remarked that there is a large deposition of subcutaneous fat, which may obscure the condition. There is no affection of the rectum or bladder.

Electrical reactions.—As far as the youth of the patient permits these are ascertained to be as follows. The most powerful faradic and galvanic currents (40 Leclanché cells), when applied to any of the nerve-trunks of the body, fail to induce any muscular contractions. The strongest faradic current also has no effect when applied directly to any of the muscles of the limbs. A moderate galvanic current to the muscles is followed by negative results, but a strong one (from 40 Leclanché cells) induces feeble contractions of a slow and tonic character A C C = K C C. No muscle is specially more excitable than another. Those of the trunk are, from difficulty of manipulation, not examined. Galvanometric experiment shows no abnormal resistance.

For six months the patient was treated with both central and local galvanisation. When seen on the 24th of April, 1883, he continues in robust general health. He appears intelligent, and although he cannot speak, he tries to do so. He has grown considerably, and has much improved in strength. He can now sit up by himself and play, and there is no tendency for the head or body to fall. The movements of the upper limbs are fair, but it is difficult to estimate their exact condition. The lower extremities move about freely, but are still weak, and the patient cannot support the weight of his body. The plantar and knee-phenomena are absent, but there is distinct evidence of the cremaster reflex. The sensibility is normal. There is now marked wasting of the muscles of the legs and thighs, and those of the arms are very soft and flabby. The

skin of the feet is cold and blue. Otherwise the condition is the same as before. The electrical reactions are changed only in this respect, that now a strong galvanic current, when applied directly to the muscles of the legs, indicates that A C C > K C C, the former being twice as vigorous as the latter.

In my judgment there can be little doubt that this also is a case of chronic poliomyelitis anterior. Whether the child has a normally developed intellect, is a question almost impossible to settle, owing to his youth, but the parents declare that they have no reason to suppose the contrary. Assuming this to be the case, this patient presents all the typical phenomena of the disease under consideration, and that of a chronic nature, as the symptoms arose in so insidious a manner that it is impossible to fix the date of their commencement. Moreover, for at least six or eight months, they gradually increased in severity, and only after that time seemed to be arrested in their further progress.

The following case is that of a girl of 14 years of age, who, however, was ill-developed for her years, and had not reached the age of puberty, so that she may be considered as a child.

III. *Case of Chronic Atrophic Spinal Paralysis in a Girl fourteen years of age.*

Augusta B., æt. 14, was first seen at the Westminster Hospital on the 10th of October, 1882. The patient is stated to have been in good health till about five months ago. Without any illness or apparent cause, she then for the first time noticed a slight weakness of her legs. This slowly increased, so that in two months she had much difficulty in walking. For the last eight or ten weeks she has been unable to stand on her feet, but has been compelled to crawl about on her hands and knees. The general health has throughout been good, there has been no pain nor distress of any kind, and the entire body above the lower extremities is perfectly normal.

At present the patient is a healthy-looking girl. She has no complaint of any kind except inability to use her legs. There is no abnormal condition of the spine, and no rectal

nor vesical disturbance. The patient cannot stand or walk unsupported, but with a little assistance she can manage to shuffle across the ward. Her gait is very feeble, the legs sink under the weight of the body, the knees are crossed over one another, and the patient treads on the outside of her feet. When lying on her back in bed, the movements of the thigh on the trunk can be performed, but they are obviously defective. The actions of the knee-joints are slow, weak, and jerky. The movements of the feet and toes are very weak and imperfect on both sides. When at rest, the knees point inwards, as well as the feet and toes, the last being in a condition of equino-varus. There is no trace of rigidity of the muscles or joints, which, on the contrary, are soft and flaccid. The plantar reflexes and knee-jerk are absent on both sides. The sensibility is everywhere normal, and there is no pain in the back or limbs. The muscles of the thighs are very thin and spare, and those of the calves and peroneal regions are markedly atrophied on both sides. The skin of the legs is cold and cyanotic. The remainder of the body is normal.

Electrical reactions.—Above the umbilicus these are normal. Below this level all the nerve-trunks respond very feebly to the strongest *faradic* currents, and to those below the knee there is no response whatever. A strong *galvanic* current to all the nerve-trunks of the lower limbs causes contractions, but these are imperfect and below the normal vigour. When the current is applied directly to the muscles of the thighs, the contractions are vigorous, K C C > A C C, but the latter is increased. To the flexors and extensors of the feet and toes the response to galvanism is diminished in quantity, and A C C = K C C, the contractions being slow and tonic.

May 20th, 1883.—The patient was seen to-day. She has been at home for some months without any treatment. She is in much the same state as regards her powers of locomotion and the other conditions of her limbs as that already described when she entered the hospital. There has been no opportunity of testing the electrical reactions.

In this patient there can be little doubt that we have a typical clinical picture of the train of symptoms resulting from chronic disease of the anterior cornua of the cord.

In addition to the three cases above detailed, I am convinced that I have seen others in children of a similar nature, although I am not at present in a position to give an account of their condition in a sufficiently extended manner to establish the diagnosis, and, moreover, a description of them would only be a repetition of the same facts. I have also met with several cases of a more local paralysis which was probably due to disease of the spinal grey matter, which, however, could not be detailed here, as, in the absence of a post-mortem examination, there always remains a doubt as to the central origin of the disease. At this moment I have under observation a child of three years old suffering from weakness of one arm, with wasting of the muscles, and no loss of sensibility, which came on gradually in a progressive manner, and which to electrical investigation presents all the conditions already enumerated to both nerve and muscle. I regard this as a limited poliomyelitis, as the paralysis occupies the entire limb, and not, as in peripheral palsy, a single nerve or the muscles which it supplies. There is not *à priori* any reason to suppose that this disease should be peculiar to adults, and specially absent from young subjects. Is it not rather more probable that hitherto it has been overlooked or confounded with other affections? As the more precise methods of modern investigation become more widely spread, as electrical and other physical tests are more universally employed in the diagnosis of nervous lesions, it will doubtless transpire that the disease under discussion is not so rare as is at present supposed.

To arrive at a correct conclusion, as to the presence of atrophic spinal paralysis, is only a question of accurate diagnosis; and to the neurologist who has been initiated into all the modern methods of investigation, this is as a rule not a difficult task. For this purpose he must not depend on any special or characteristic symptom alone, but must fully consider all the circumstances of the case and bring to bear on the subject all the critical enquiry and armamenta of physical research which he possesses. Poliomyelitis, in its chronic form, if we except lead palsy, which it is improbable would occur in a very young child, is not likely to be taken for any other spinal disorder except progressive muscular atrophy, which affection, according

to Duchenne, is not uncommon in children. In both these diseases the anterior cornua are degenerated, though at present we have no reliable pathological distinction between the two. But the clinical sequence of symptoms in each case is so different as to warrant us in regarding them as separate affections. In chronic atrophic paralysis the parts are attacked "en masse," while in progressive muscular atrophy they are affected piecemeal. In the former the paralysis appears first, and the atrophy of the muscle follows; while in the latter there is no real palsy, but the weakness is in proportion to the loss of muscular substance. In the first, the nerves, both at their trunks and peripheral extremities, are degenerated; in the last they remain for long intact, hence the marked difference in the electrical reactions. Finally, in poliomyelitis the reflexes are abolished; while they are present till a late stage in progressive muscular atrophy. Why degeneration of the anterior cornua should, without perceptible anatomical difference, cause two diseases unlike one another, we do not yet know, and it remains for future enquirers to determine the special anatomical distinction in such case. The fact remains that what appears to be the same morbid process is followed by two clinical conditions, not only unlike in symptoms, but dissimilar in progress and termination.

It is certain that, in the distinction between atrophic spinal paralysis and progressive muscular atrophy, the most important agent which we possess is the electrical test. Not only is this a means of determining the difference between these two diseases, but it is the most rapid and certain method of distinguishing poliomyelitis from many other doubtful and obscure paralyses. In children there may be many forms of weakness due to a variety of causes, such as brain disease, compression of the cord, simple emaciation, &c., which are often confounded together, and which it may sometimes be difficult or almost impossible to differentiate. The electrical test alone gives us the most reliable and definite information in distinguishing these and other conditions from that paralysis which is due to degeneration of the anterior cornua of the cord.

When the poliomyelitis is limited, and the paralysis result-

ing therefrom is confined to a limb or part of a limb, the diagnosis is sometimes very difficult. It is then far from easy to determine whether the symptoms are due to a cord or to a peripheral nerve lesion. The same general rules already referred to must be considered. In addition, the fact of the paralysis being confined to the trunk of a single nerve, or distributed more profusely, the condition of the sensibility and the history of the case, will further assist us in the solution of this delicate problem.

The electrical reactions in the three cases above detailed were definite and characteristic, and accorded in every particular with those found in acute infantile paralysis, and in the chronic poliomyelitis of adults. There was great diminution or total loss of response to both the faradic and galvanic stimuli when applied to the nerve-trunks of the affected parts. The muscles, when directly irritated with the induced current, also failed to respond, but galvanism to these caused contractions more or less vigorous, of a slow and tonic character, with the polar phenomena altered or reversed—in short, the typical "reaction of degeneration." In all the cases of poliomyelitis which have come under my notice, this condition, or a slight modification of it, has existed, namely alteration in the excitability of both nerve and muscle. In Professor Erb's case, the conditions were somewhat different. Here, when the nerve-trunks were stimulated with both currents, there ensued practically normal contractions, and the same took place when the muscles were directly irritated with faradism. With galvanism, on the other hand, the muscles displayed tonic contractions with inverted polar reactions, the whole constituting what has been termed "the middle form of the reaction of degeneration." I have not met with this condition in poliomyelitis, as in all the cases which I have investigated the nerves were distinctly altered. If the last condition was universal, the test would serve to distinguish this disease from progressive muscular atrophy, in which the nerves are generally normal. The case under notice cannot have been an example of this affection, as it rapidly recovered, and the reflexes were absent. At the same time I consider that the electrical reactions described were not those usually

met with in either acute or chronic poliomyelitis, but rather those seen in progressive muscular atrophy. A possible explanation of the unusual phenomena in this instance may be that here the disease was ill-developed or slight, as evidenced by its subsequent rapid and complete recovery. The lesion of the nerves was sufficient to cause certain peculiarities in the electrical reactions (the partial reaction of degeneration and the sluggish contractions), but not to render them altogether insensitive to stimulation, as indicated by muscular contraction following indirect irritation. These facts are of prognostic value, as both Erb and Kast have suggested, pointing to a mild progress of the disease and a favourable termination.

An imperfect development of the disease may also explain the presence of the peculiar phenomena described in Professor Erb's case, following both direct and indirect excitation by the two currents, namely the great tonicity of the contractions. I have seen this in certain cases of muscle-degeneration, but never, as far as I can recollect, in poliomyelitis. In this, as in other deviations from the usual type, we are not yet aware of its exact clinical significance. I have occasionally observed another variety of altered reaction,[1] the explanation of which is equally obscure. In certain diseased conditions of muscle, a rapidly interrupted galvanic current causes a sudden and vigorous contraction, immediately followed by relaxation, although the current continues to flow. In a healthy muscle this would of course be accompanied with tetanus during the application of the electrode, whereas in the above instance the contraction is momentary. This condition I have seen in peripheral paralysis,[2] lead poisoning, &c. It would seem as if in certain forms of muscular degeneration without complete disorganisation, the shock of the interrupted galvanism causes contraction, but that the muscle, owing to disease, has lost its tonicity and capacity to maintain a tetanic spasm.

Those modified electrical reactions, those deviations from the usual types, of which we are at present ignorant of the full significance, will doubtless, as our experience extends, prove valuable agents in the diagnosis and prognosis of disease.

[1] 'Electro-diagnosis,' p. 73. [2] *Loc. cit.* p. 139.

ON INFANTILE SPASMODIC PARALYSIS.

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THE cases which I am about to describe in this paper possess one striking and characteristic feature. Whatever may be their pathology—and on this point there is ample scope for discussion—they are all linked together by one clinical bond. I refer to the condition of the lower extremities.

Although in many cases other symptoms exist, which when rightly considered assist greatly in the interpretation of the clinical phenomena, a peculiar attitude of the legs and a characteristic gait are common to all.

The lower extremities are more or less flexed at the hip and knee-joints, the thighs are rigid and adducted, the knees in contact, the legs inclined outwards, and the heels often drawn up from the ground by the contraction of the gastrocnemii. Briefly, this condition is one of spastic paraplegia. But in the child, adductor spasm seems to prevail; whereas in the adult extensor spasm is commonly observed.

The cases which have come under my own observation may be divided broadly, and for the sake of convenience, into two groups.

1. Cases of spastic paraplegia, in which certain nutritive changes are present in one upper extremity.

2. Cases in which the lower limbs alone are affected.

It must be mentioned that several instances of all four extremities being involved are also on record, but this was not present in any of my own cases. I shall, however, take the opportunity, when discussing the nature of the disease, to allude to this condition.

Of the first group I have but two cases to relate. In one[1] the patient was a boy, aged 13, whose family are free from all neurotic taint. He has been subject to fits, occurring at intervals of from one to five months, or more, since he was a year old.

When a child, three years of age, he had a fall, since which (according to the friends' statement) he has been paralysed in both legs and right arm. The fits are preceded by irritability of temper, and by a distinct aura, consisting of a "nasty taste" in the mouth.

Movements, with trembling, then begin in the right hand, and rapidly extend over the rest of the body.

During the attacks he bites his tongue and foams at the mouth. Afterwards, he is drowsy, dull, and sometimes so weak that he is confined to bed for four or five days.

With the exception of chicken-pox, and the presence of worms when he is constipated, the patient has suffered from no other complaint.

The boy was admitted into St. Thomas's Hospital, under Dr. Bristowe, on account of a severe epileptic attack.

When first seen, he was strongly convulsed, and there were clonic spasms of the hands, especially the right. The upper extremities were far more affected than the lower, which, with the exception of a few slight twitches, were motionless. The muscles of the face and eyeballs were strongly convulsed; the pupils constantly varied in size. The patient was quite unconscious. The patellar reflex was said to be entirely absent, although there was marked ankle-clonus.

On subsequent examination, the patient was found to be a fairly intelligent boy. Although his friends consider him somewhat deficient mentally, he reads, counts, and remembers well. His expression is rather vacant, but there is certainly no notable intellectual deficiency. The pupils are equal and act well, both to accommodation and to light. There seems to be slight lateral nystagmus. The tongue is protruded straight, and there is no facial paralysis. The right upper extremity is much smaller than its fellow, the ulna being an inch shorter than

[1] "An Anomalous Case of Infantile Hemiplegia;" 'British Medical Journal,' Feb. 18th, 1882.

the bone on the opposite side. The muscles are considerably wasted, and Dr. Kilner, who examined them electrically, said they all present the reaction of degeneration. The fingers are kept apart, hyper-extended at the metacarpo-phalangeal joints, and semiflexed at the other joints. This condition is especially marked in the index and middle fingers. There is very pronounced loss of power both of the arm and hand. The tendon-reflexes are much more evident than on the left side.

The left upper extremity is in every respect natural.

The legs are rigid, and there is well-marked talipes equinus on both sides. The right lower extremity is decidedly stiffer than the other, and the contraction of the gastrocnemius on the left side is not so great as on the right, since he can, although with some little difficulty, bring the left heel to the ground. The patellar reflex is exaggerated, but there is no appreciable difference between the two limbs in this respect. Ankle-clonus was usually obtainable on both sides to nearly an equal extent, but on the last occasion when it was tried it existed on the right side only. Occasionally it was impossible to provoke it, in consequence of the great rigidity of the calf-muscles.

The lower limbs are not atrophied, the electrical reactions of the muscles are normal, and there is no loss of power.

The plantar reflex is present on both sides, but it is difficult to say whether it is exaggerated or not.

The abdominal reflex exists on the left side only. The right cremasteric reflex cannot be obtained in the usual way. On stimulating the inner aspect of the left thigh, both testicles are simultaneously retracted.[1]

Sensation is everywhere intact, and there is no loss of control over bladder or rectum. There is no atrophy of the thoracic wall or pelvis on either side.

But the most striking feature in this patient is his gait. When asked to walk, he supports himself for a moment by leaning against some object. It is evident, indeed, that,

[1] I had the opportunity of examining this boy since the above was written. The plantar reflexes are decidedly exaggerated, the right being the most marked. On stimulating the inner side of the right thigh, I succeeded in obtaining a slight reflex movement of the corresponding testicle.

although not lacking the muscular force, he is unable to maintain his equilibrium, when upright, without extraneous aid.

As he stands, he is seen to rest on the balls of the toes, the heels are drawn up from the ground, the hip and knee-joints are slightly flexed, the thighs are adducted, the knees almost in contact, the legs incline outwards, and the feet are much inverted. During progression, he brings each lower limb forwards by describing an arc with the convexity outwards. At the same time the head and shoulders are thrown back, the dorsal curve is greatly exaggerated, and the right arm, which is flexed on the forearm, with the corresponding hand dropped on the wrist, is brought up nearly to a level with the chin. As he walks he keeps the right upper extremity in this position; but the opposite limb is moved backwards and forwards, and, together with the trunk, which sways powerfully from side to side, serves to maintain the equilibrium during progression.

The condition I have just described depends, I believe, on a cerebral lesion, although the secondary spinal effects tend to obscure the true nature of the disease.

The fact that the epileptic attacks began in the right hand and affect the corresponding side more powerfully than the other limbs, strongly suggests that the lesion is primarily cerebral. The atrophic condition of the right upper extremity and the complete immunity of the left upper limb point to the unilateral nature of the lesion.

If the state of the lower extremities alone were considered, it might be urged with much force that the disease was spinal. It must be mentioned, however, that although both limbs are extremely rigid, the right is slightly more affected than the left. The absence of the right abdominal and cremasteric reflex is an additional argument in favour of the unilateral cerebral nature of the affection. Rosenbach and Jastrowitz have pointed out that these reflexes are lessened or abolished on the paralysed side in the case of disease of one cerebral hemisphere.

It is curious that, although the right cremasteric reflex could not be obtained by stimulation of the inner side of the thigh on the corresponding side, the phenomenon was called

forth by irritation of the skin on the opposite side, and that the reflex movements were apparently synchronous. This fact seems to indicate that the motor cells in the lumbar region are more intimately connected on each side than is usually the case.

I believe that in this patient there exists some lesion or congenital defect in the motor part of the left cerebral hemisphere. This lesion, whatever may be its nature, has been followed or accompanied by change in both lateral columns, and it is highly probable that the motor cells which control the movements of the right upper extremity have undergone atrophic changes, or have been arrested in the natural course of their development.

Why, it may be asked, have both lateral columns been attacked? Cases of hemiplegic rigidity in children are not uncommon, but it is rare to find both lower limbs affected in consequence of a unilateral cerebral lesion. In a paper about to appear, I have given some cases of paraplegic rigidity in hemiplegia and have discussed their probable nature.[1]

These cases may be explained as follows. It is well known that the decussation of the bulbar pyramids is liable to great variations. The direct pyramidal tract usually bears a very small proportion in the number of its fibres to the crossed tract, but occasionally the order of things is modified or absolutely reversed.

Now, the fasciculi of Türck, which are concerned with the transmission of voluntary motor impulses, diminish in number as they pass down the cord, generally disappearing about the middle of the dorsal region.

It seems to me possible that these fibres dip into the cord and pass downwards on the same side, to enter into relation with the multipolar cells in the lumbar region. It is a matter of common knowledge that one cerebral hemisphere controls not only the movements of the opposite limbs, but to a certain extent of those on the same side.

It is quite conceivable that when the non-decussating pyramidal fibres are unusually numerous, the loss of power might be distributed between both lower limbs, and not

[1] "On Paraplegic Rigidity in Hemiplegia;" St. Thos. Hosp. Reports for 1882.

practically limited to one leg only. Cases of this nature are not very uncommon.

Charcot believes that paraplegic weakness or rigidity supervening in hemiplegia depends on a second decussation of the motor fibres in the dorsal region. In other words, he suggests that there is not only a decussation of the pyramidal tract-fibres at the medulla, but a partial and variable intercrossing of the same fibres in the spinal cord.

The atrophy of the right upper extremity is worthy of remark. It depends probably on the fact that the spinal nerve-cells which preside over the movements of the upper limbs are much less intimately connected across the cord than those which control the movements of the lower limbs.

Muscular atrophy in lateral sclerosis, whether primary or secondary, must be looked upon as exceptional. When it does occur, as in late hemiplegic rigidity and amyotrophic lateral sclerosis, the arm is most liable to be affected.

It would seem as though bilaterally associated cells were mutually protective, to a certain extent, against the extension of influences affecting their nutrition.

The next case is that of a young man, aged 19, who was admitted into St. Thomas's Hospital under Dr. Bristowe in the early part of this year. He was a poorly-nourished youth, of about average height, with decided prominence of eyeballs, but without any enlargement of the thyroid gland. The patient was born at full time and was one of twins. The birth was difficult, but no instruments were used. The other child was born dead.

According to the friends, the patient was born head first.

There was no history of fits or of any hemiplegic condition in infancy.

Both his father and mother had died of phthisis, but there was no history of nervous diseases in the family, and, as far as could be ascertained, there was no suspicion of congenital syphilis.

He began to walk when between four and five years old, and from the first walked on his toes. Later, he walked more on the soles, but about two years ago he again took to walking on his toes.

About this time he had pains in both ankles, which of late years became worse, the right hip being also affected.

On admission, he complained of weakness of legs and frontal headache.

His gait presented the same characteristics as that of the patient whose history has just been given.

As he walks, he brings one foot and leg in front of the other, and as the latter is being brought forward in its turn, the knees overlap and rub. The feet are not applied well to the ground, but rest almost altogether on the balls of the toes.

During progression, there is some lordosis, the shoulders are thrown back, and he waddles from side to side. When lying down, the legs are more or less adducted and stiff. The rigidity becomes more marked when passive movement is attempted. In the supine position the lordosis disappears, and there is no ankylosis of the hip-joints.

Both knee-jerks are very brisk, but the gastrocnemii are so contracted that ankle-clonus is only obtained occasionally, and with some difficulty.

Sensation is nowhere absent; but below the knees it seems to be a little deficient. There is no affection of bladder or rectum. The left hand is curiously deformed; the middle, ring, and little fingers are flexed at the first interphalangeal joints, and he cannot extend them. The ends of the fingers and thumb are bulbous, and the epidermis over them is very thick and hard. The nails are imperfectly developed, being small, thick and claw-like.

In fact, the terminal phalanges are much atrophied. The fingers of the left hand sometimes become more contracted, and the arm feels stiff. The hand and arm are slightly weak. Sensation is said to be a little blunted in the left arm.

The right upper extremity is quite natural, the elbow-jerk is present equally on both sides. The abdominal, cremasteric and plantar reflexes are also obtainable on both sides. The special senses are unaffected, and there is no paralysis of face or tongue. The patient is fairly intelligent, but has never given much time to school-work. All that can be said of him is that he is dull and somewhat slow of comprehension. The chest is a little deformed, the sternum being rather prominent

below. There is no asymmetry of the skull, and no abnormality of palate.

The measurement beneath axilla and over shoulder is 15¾ inches on right side, and 15½ inches on left side.

The greatest circumference of right arm is 10 inches; of left, 9½ inches.

The length of right arm is 14 inches; of left, 12½ inches.

The length of each forearm is 11 inches.

The circumference of right palm is 8¼ inches; of left, 7⅞ inches.

There is no difference in the measurements of the lower extremities.

To sum up, the left arm is 1½ inch shorter than the right, and the circumference of the left hand nearly an inch less than the right.

During his stay in the hospital his sensation was several times tested and found natural, although on the first examination some deficiency was thought to exist below the knees and in the left arm. When first seen, he could only walk a few yards, as he soon staggered and fell. He improved considerably in this respect after a few weeks, but the peculiarity of the gait remained, the knees knocking together and overlapping, and the foot being swung round with a semicircular movement.

The nature of this case is even more obscure than the other, but the involvement of three limbs makes me inclined to think that the cause is primarily cerebral. The absence of fits, on the other hand, is an argument against this presumption.

The ill-developed condition of the terminal phalanges of the left hand was new to me, but I have lately seen a similar affection in a patient who is suffering from dislocation of both wrist and shoulder-joints. There is no suspicion of ataxy, but there is evidence of some degeneration in the lateral columns of the cord.

I will now pass on to the second group of cases. I take the opportunity of expressing my indebtedness to Mr. Clutton and Mr. MacKellar, of St. Thomas's Hospital, who kindly called my attention to some of these cases. I must remark too, incidentally, that patients of this class are to be found

more especially in the surgical out-patient department, where not unfrequently the true nature of the disease is overlooked.

Case I.—Male, æt. 9. The father died of phthisis, and one brother in infancy, of cause unknown. There is nothing else to note in the family history. The mother states that he was not born prematurely. The labour was difficult, and instrumental aid was necessary.

He had convulsions for nearly a month after his birth, and they continued at intervals until he was a year old. No loss of power in one arm and leg was ever noticed. He has never walked properly, but began to step when about two years of age. He has never been to school, so is very backward.

The patient is a thin, pale, half-idiotic looking boy, with a large but symmetrical head, a low and retreating forehead, drooping eyelids, prominent and overlapping front teeth.

He can neither spell nor add up, he laughs and cries readily, and is very dirty in his habits, passing his evacuations under him. His condition, there is no doubt, is one of partial idiocy.

He can speak simple words and understands easy questions.

When lying down, the knees, which are slightly bent, are in contact; the thighs are adducted and nearly touch. The feet are extended, the right partially overlapping the left; the great toes are crossed, and the heels are turned outwards. He can neither bend nor extend the feet, but there is fair movement at the knee and hip-joints.

The plantar reflex is absent on both sides.

The knee-jerks are much exaggerated.

On striking the tendon the leg at once becomes extended, and remains so for a few seconds trembling, before falling back into its usual position.

Ankle-clonus can be obtained on both sides, and there is occasional knee-clonus.

The measurements of the two legs are about equal.

The abdominal, cremasteric, inter-scapular and gluteal reflexes are brisk, but not more marked on one side than another.

He can neither stand nor walk without help. When he

tries to go forward he slides the foot along the ground with a semicircular movement.

The gait and the spastic condition of the lower limbs are the same as in the cases already described.

The arms are in every respect natural.

The terminal phalanges both of fingers and toes are well developed.

There is no affection of common or special sensation. The chest wall is of good shape, and both thoracic and abdominal organs appear healthy.

Case II.—Male, æt. 8. The mother is said to have died from "asthma." The birth of the patient occurred at full time, the head presented, and delivery was quite easy.

The child was healthy up to the age of twelve months, and had cut several teeth. He then had about six fits, extending over a period of as many months. After these attacks, his friends noticed that there was something wrong with his eyes. At the age of three he had not begun to walk, and it was thought that the legs were "queer."

On examination the child is seen to be fairly nourished and slightly undersized. The forehead is prominent, the upper part of skull large and square-shaped, in marked contrast to the face, which is small. There is no asymmetry of skull and no malformation of palate.

The child is really an idiot. He is never still, always singing, is very mischievous, and peevish, and swears at the slightest provocation. He is dirty in his habits, and passes the urine and fæces under him.

The condition of the eyes is said to date from the time of the fits.

There is double internal squint, marked lateral nystagmus, and inequality of pupils, the right being the larger. The gait presents the peculiar characteristics seen in the other patients. He seems to be a little flat-footed. The knee-jerks are very brisk, but there is no ankle-clonus.

The arms are quite natural. Sensation is unimpaired. The chest is well formed.

Case III.—Female, æt. 15. There is nothing of importance in the family history.

The patient was born at the eighth month, and was not one of twins.

There was no difficulty about the birth. She has never had fits nor anything like one-sided weakness. Practically she has never walked, although she began to make an attempt at the age of four.

The patient, who is the daughter of a clergyman, is a refined and intelligent girl. There is absolutely no mental deficiency. The legs are bent on the thighs at an angle of about 150°. The tendons of the hamstrings are very rigid.

When she tries to walk with assistance, the balls of the toes drag along the ground, the legs are strongly adducted, the knees rubbing and overlapping.

The feet are kept in a position midway between extreme flexion and extension.

The toe-nails are all extremely small and ill-developed, looking as though they had broken off short.

The patellar-tendon reflex is brisk, but the range of movement is not extensive, on account of the contraction of the hamstrings. There is double ankle-clonus. The plantar reflex appears to be absent. Sensation is nowhere impaired. The functions of the bladder and rectum are quite natural. The arms are unaffected. There is slight lateral nystagmus on looking to the extreme left. The special senses are not involved.

Case IV.—Female, æt. 3. The mother has been married four years and three months, and has had three children, of whom the patient is the eldest.

The mother had a fall when six months pregnant, and is inclined to ascribe the child's present condition to that cause. Delivery took place at the end of nine months; the head presented, but the labour was tedious and instruments were used.

At the age of ten months the little girl had a convulsive attack, affecting the left side only. A month later, the right side was similarly attacked. This is the voluntary statement of the mother. She is quite certain that one side alone was affected each time.

Nothing like hemiplegia was noticed after the fits.

The patient began to walk at the age of eight months. Dentition did not commence until she was a year old.

When two years of age she began to talk, and can say anything now. The child is of about average size, fairly intelligent, not mischievous, but rather cross. No measurements of the skull were taken. The palate is somewhat high and arched. When walking, the thighs are adducted and the legs describe small circles. The knee-jerks are well-marked and apparently exaggerated. There is no ankle-clonus. There is slight talipes equinus on both sides.

As I only saw the child once, the history is somewhat imperfect; but at the time I satisfied myself that the case fell into the same category as those above described.

In addition to the four instances I have given, two or three more of similar nature have come before me. Unfortunately I have no notes of them.

What, it may be asked, is the pathological nature of these cases? Are they of cerebral or of spinal origin?

Is the lesion, wherever it may be, of an artificial or of a congenital nature?

Dr. Ross, of Manchester, has recently recorded several instances of this condition in a recent number of 'BRAIN,' and has also given the details of a post-mortem examination.[1]

In this case the arms were also affected. On each side, in the position of the fissures of Rolando, there was a deep sulcus, which extended from the point of bifurcation of the Sylvian fissure upwards to near the longitudinal fissure. "The lateral walls of each sulcus are formed by an infolding of the grey matter of the cortex, and these walls are so closely applied to each other that there is no great cavity left between them. In depth each sulcus extends the whole thickness of the centrum semi-ovale, and opens into the roof of the corresponding lateral ventricle. The opening into the ventricle on each side is only large enough to admit the little finger, and each opening is surrounded by a ring of grey matter, having all the naked-eye appearances of the cortex. . . . With regard to the fissures and convolutions, those in the

[1] "On the Spasmodic Paralyses of Infancy;" 'BRAIN,' No. 1, p. 477.

neighbourhood of the defect are radially directed towards the centre of the cavity. The ascending parietal and ascending frontal convolutions apppear to be absent on both sides."

On microscopical examination the giant cells were absent from the walls of the cavity on each side. The cells too in the inner division of the third layer were large, round, nucleated, and devoid of processes.

The anterior pyramids of the medulla oblongata were about half the natural size, and the lateral columns of the cord were also smaller than the corresponding parts in a healthy cord. It would appear that no qualitative change was found.

Dr. Ross believes that the porencephalous defects were due to an arrest of development, and not to any destructive process.

Mr. Hutchinson has recorded a case which in many respects resembles the preceding. At his request I saw the child, and I am inclined to think that the condition is closely allied to that of the patients I have described.[1]

From a perusal of several cases of spastic paraplegia in children, it would appear that in all probability they depend on some congenital defect in the nerve-centres.

In connection with this subject it is worth while to recall the researches of Parrot and Flechsig on the development of the pyramidal tracts. The motor fibres take origin from the ganglionic cells in the cortex just below the fissure of Rolando. The process begins soon after birth, but is not completed until the end of the first year or later. The fibres gradually descend, by a kind of budding, into the crus, pons, medulla oblongata, and spinal cord.

Assuming a double porencephalous defect near the fissure of Rolando, such as Dr. Ross has described, it might happen that the downward passage of the pyramidal-tract fibres is arrested high up in its course. In that case both arms and legs would be involved. If the arrest took place at a lower level, the legs alone would be affected. This hypothesis is partly supported by the post-mortem examination given by Dr. Ross. Here the pyramidal tracts were not diseased, but simply small. It is well to bear in mind, however, that the

[1] "A second Report on a case of Double Hemiplegia of Infants, with Idiocy and a Transitory Dermatitis." 'Trans. of the Path. Soc. of London,' vol. xxxiii.

pathological anatomy of simple spastic paraplegia in children is practically unknown.

The frequent occurrence of fits, mental deficiency, and other cerebral symptoms suggests that the condition of the legs is either dependent on, or accompanied by, some lesion in the brain.

A satisfactory explanation of the two cases in the first group is somewhat difficult.

In both there was a trophic lesion in one arm, the other arm being absolutely unaffected.

Accordingly I am inclined to believe, as I have already said, that the lesion is primarily unilateral in the brain, and that the paraplegic distribution of the rigidity is due to an unusual decussation of the pyramidal-tract fibres. The cases are undoubtedly rare. I have seen no similar recorded instances.

After carefully studying my own cases, and comparing them with those of Dr. Ross and Dr. Gee,[1] I came to the following conclusions.

There is nothing special in the family history.

No condition, either during pregnancy or during birth, has any influence.

Convulsive attacks in infancy occur in nearly half the cases.

The first fit usually takes place during the first year, sometimes shortly after birth, more rarely at the second year of life. As a rule the attacks extend over a few days or even months, and then cease either completely, or recur after an interval of months or years.

The fits vary from one or two to half-a-dozen or more; sometimes they are very numerous. Unilateral attacks are rare. In one of my own cases the left side was first attacked and then the right, but on each occasion the seizure was purely one-sided. In one of Dr. Gee's cases the left side was chiefly affected, and a temporary weakness was noticed afterwards. In one case internal strabismus and nystagmus were said to follow shortly after the fits. In one case only an aura and local commencement of the fits existed.

[1] St. Bartholomew's Hosp. Reports, vols. xiii. and xvi.

Walking is nearly always delayed. Sometimes the patients never walk. The attitude of the lower limbs is common to all the cases, and if progression be possible, the gait is the same in all.

Dentition is late. Articulation is often quite natural. Hesitation and drawling have been observed. Intelligence is natural in a large number. Mental deficiency, varying from absolute idiocy to mere dulness, is not unfrequent. Ocular symptoms occur in a considerable proportion—nearly a half. Converging strabismus is the most common; nystagmus with or without decided squint less frequent; inequality of pupils and protrusion of eyeballs rare.

The tendon reflexes are well marked, probably exaggerated, in all the cases. Ankle-clonus exists in many, but is sometimes difficult to obtain. The skull is generally natural, rarely unsymmetrical. Occasionally it resembles that of a microcephalic idiot.

Among occasional symptoms are deformity of chest, choreiform movements, cramps, arching of palate, and weakness of back.

Muscular wasting and the reaction of degeneration are unrecorded, except in one of my own cases. Sensation and the functions of the bladder and rectum are unaffected.

A word as to treatment. It is clear that drugs can be of no avail. In Case I. of the second group the tendo Achillis on each side was divided by Mr. Clutton. The result was discouraging, as might be expected from the nature of the affection.

THE PULMONARY PATHOLOGY OF GENERAL PARALYSIS.

BY J. CRICHTON-BROWNE, M.D., LL.D., F.R.S.,

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EVERY one who has had to make *post-mortem* examinations of the bodies of persons who have died of general paralysis of the insane must have been struck by the fact that the pathological changes in that disease are not confined to the brain and nervous system. Having their primary seat and permanent home in the cerebral cortex, these changes spread in various directions, and send forth their evil influences throughout the system, so that when the disease is far advanced, there is scarcely an organ or tissue that does not present some morbid modifications. The essential *substrata* of the diseased processes in the central nervous system must, of course, always receive a principal share of attention; but the secondary degenerations of other parts are certainly deserving of some consideration, both because they reflect light on the conditions in which they originate, and because they may alter or cut short the course of the malady to which they are themselves subsidiary.

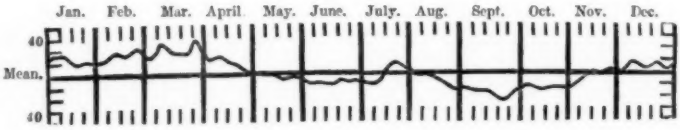
Among the secondary degenerations in general paralysis those that affect the lungs are not the least conspicuous and important. In a large majority of the cases of this disease in which autopsies are performed, coarse changes in the pulmonary structure are plainly visible, and in many instances these are of a character which indicates that they have interrupted the progress of the cerebral disorder, and cheated its victim of the dregs of life.

But although thus manifest and significant, the pulmonary changes in general paralysis have hitherto received only a

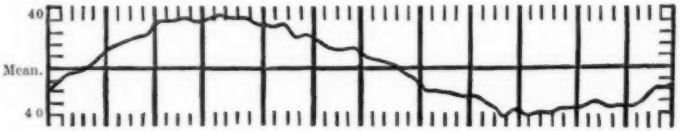
passing notice. They have not been subjected to careful study or analysis, and it would be impossible to find on record to-day any definite information as to the relative frequency of their different varieties. Under these circumstances, it has seemed to me that it might be useful to sum up a few observations which I made upon these changes some years ago. The observations are somewhat superficial, and, being simply macroscopical, have not the value which they would have possessed had they been supplemented by histological investigations; but still even in their crude state they are not perhaps without interest and practical suggestiveness.

My observations were carried out in the West Riding Asylum, and were made on the bodies of 100 patients—80 males and 20 females—who died in that institution of general paralysis between April 1872 and August 1875. The average age of these patients at the time of death was 41 years and 7 months in the men, and 42 years and 5 months in the women; and the average duration of asylum treatment was 16 months and 2 weeks for the men, and 24 months and 1 week for the women. The earliest age at which death from general paralysis occurred was 24 years in a man, and 32 years in a woman; and the greatest age at which it occurred was 55 years in a man, and 57 years in a woman. The average age for the incursion of the disease is about the same for both sexes, viz. 40 years; but the advance of the malady is more rapid and violent in men than it is in women. In the 100 cases which are here reviewed, all cases of general paralysis in the West Riding Asylum that reached a fatal termination, within the period defined (April 1872 to August 1875), are included, whether death happened in the ordinary course of the disease, or in consequence of some intercurrent malady; but it is to be noted that in this group of cases intercurrent maladies did not appear until towards the natural close of the general paralysis. In every case the patient was in the fourth or last stage of the disease when death took place, and in a large number of cases that stage was so far advanced, that the intercurrent malady might be looked on rather as a mode of death than as a fatal complication. And yet evidence is not wanting that

intercurrent affections, although so late in intruding in these cases, did to some extent shorten their duration. Of all such affections, those of the respiratory apparatus are the most frequent and deadly, and if we examine the distribution throughout the months of the year of the mortality in the cases which we are considering, we shall at once recognise the influence of diseases of the respiratory organs. General paralysis is essentially a nervous disease, and, like all other nervous diseases, it is most fatal in the spring months. Were the mortality which results from it, in a large number of cases, when it runs an uncomplicated course, arranged in the manner adopted by Dr. Arthur Mitchell and Mr. Buchan, in their masterly investigations into the influence of weather on health in London, it would be found that the curve which could be regarded as its constant would closely correspond with the curves of convulsions, epilepsy, whooping-cough, and other nervous diseases. In the accompanying woodcuts reproduced from a paper by Mr. Buchan, the curves for convulsions and whooping-cough are given, the straight black line in each figure being drawn to represent the mean weekly death-rate on an average of the fifty-two weeks of the year, and the figures on the margin the percentages above or below the average.



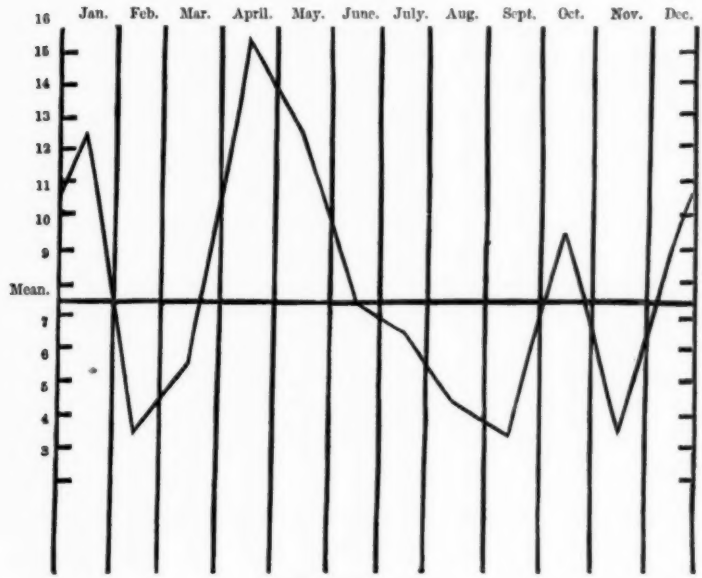
CONVULSIONS.



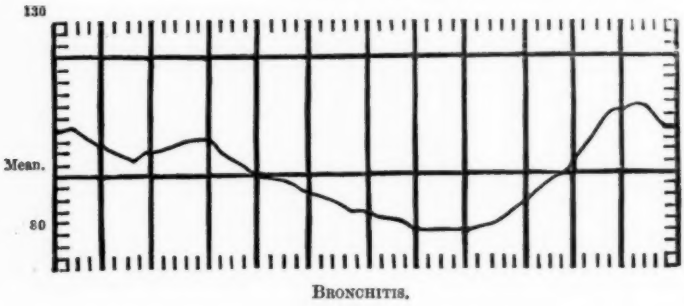
WHOOPING COUGH.

It will be gathered from these woodcuts that the maximum mortality from convulsions and whooping-cough occurs in

the spring months—February, March, April, and May; and that their minimum mortality is in the autumn months—August, September, and October.



General Paralysis.



Bronchitis.

Now if the incidence of the mortality from general paralysis in the several months of the year be thus diagrammatically represented as regards the cases which we are examining, and

which, although too few in number to justify any very precise conclusions, may yet supply some trustworthy indications, we shall see that this disease too has its maximum mortality in April and May, and its minimum mortality in August and September; but that its curve likewise presents a second very decided rise in December and January, in this respect resembling pneumonia and bronchitis, which have their maximum mortality in the cold months of the year.

When the curve of the mortality from general paralysis can be constructed from a number of cases so large as to obviate mere accidental variations, such as that in the November fall in the accompany chart, it will be found that it is really a compound of the curves of the mortality from nervous and respiratory diseases, and it may hence be inferred that the affections of the respiratory organs which complicate general paralysis have a certain independent influence on its course and termination.

The first point to which I directed attention in inquiring into the pulmonary pathology of general paralysis was the weight of the lungs, and the results obtained with reference to that subject are exhibited in Table I.

The mean weights of the lungs here given—26½ oz. or 756 grammes for the right, and 24 oz. or 682 grammes for the left in the male, and 18½ oz. or 526 grammes for the right, and 15½ oz. or 438 grammes for the left in the female, are so greatly in excess of all standard lung-weights, that it is obvious that they have been raised in the case of general paralytics by morbid conditions of the lungs, the influence of which is also apparent in the enormous difference between the highest and lowest weights, as shown in the Table. In order to determine to what extent the increase in the weight of the lung in general paralysis was due to pathological changes, I selected from the 100 cases all those in which the lungs were normal, or at least free from coarse and obvious pathological change, and calculated their weight separately. These cases were sixteen in number, including fourteen men and two women, and in them the lungs were practically sound, no departure from healthy structure being observed by the unaided eye beyond a slight emphysema at the margins in one

TABLE I.

SHOWING THE WEIGHT OF THE LUNGS IN ONE HUNDRED CASES OF GENERAL PARALYSIS.

——		Right Lung.						Left Lung.					
		Highest.		Lowest.		Mean.		Highest.		Lowest.		Mean.	
		Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.
Persons .	100	46·75	1326	7·50	214	25·00	710	56·50	1600	10·50	298	22·25	633
Males .	80	46·75	1326	13·00	369	26·50	756	56·50	1600	12·25	347	24·00	682
Females .	20	31·00	879	7·50	214	18·50	526	22·00	624	10·50	298	15·50	438

or two cases. The weight of the lungs in these cases is given in Table II.

On comparing this Table with that showing the weight of the lungs in all the 100 cases dealt with in this paper, it is seen that the aggregate effect of whatever pathological changes occur in these organs during general paralysis, is to increase their average weight by about 2¼ ounces, or 122 grammes, in the male sex, and by about 1 ounce, or 27 grammes, in the female sex. A comparison, however, of the weight of the normal lungs in general paralytics, with the weight of the normal lungs in persons not affected by that malady, shows that during the course of uncomplicated general paralysis the lungs tend to diminish in weight. According to Dr. John Reid's observations, the average weight of the right lung is 24 ounces in the male, and 17 ounces in the female; and the average weight of the left lung is 21 ounces in the male, and 15 ounces in the female. But the average weight of the right lung in the cases included in the Table here submitted, is 22¼ ounces in the male, and 17½ ounces in the female; while the average weight of the left lung is 19½ ounces in the male, and 15 ounces in the female. In female general paralytics it would seem that the lungs are, as regards weight, fully up to the healthy standard (in the case of the right lung the weight given in this Table exceeds that given by Dr. John Reid by half-an-ounce), but it is to be noted that the average quoted is derived from the weight of the lungs in only two female general paralytics, who were both considerably above average height, and cannot, therefore, be regarded as trustworthy. In male general paralytics, however, the diminution in the weight of the lungs is very decided, amounting to an ounce and three-quarters, on an average, in the right lung, and an ounce and a half in the left. This loss of pulmonary substance in general paralysis, when no actual disease has invaded the lungs, must be regarded as in some degree analogous to the atrophy which is seen in advanced age. Senile changes are indeed anticipated and exaggerated in general paralysis, in which a general failure of nutrition is witnessed in muscles, bones, viscera, and indeed in every organ and tissue. An abnormal trophic innervation is doubt-

TABLE II.

SHOWING THE WEIGHT OF THE LUNGS IN SIXTEEN CASES OF GENERAL PARALYSIS, IN WHICH THESE ORGANS WERE NORMAL.

		Right Lung.						Left Lung.					
		Highest.		Lowest.		Mean.		Highest.		Lowest.		Mean.	
		Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.
Persons .	16	30·00	851	15·25	430	21·75	617	27·50	780	14·00	400	18·75	535
Males .	14	30·00	851	15·50	440	22·25	634	27·50	780	14·00	400	19·50	551
Female .	2	20·00	567	15·25	430	17·50	499	15·25	430	15·00	425	15·00	428

less responsible for much of the impaired nutrition that is seen in this disease, but in the case of the lungs it is scarcely necessary to resort to any trophic theory to account for some wasting in them. It is to be remembered, that in the latter stages of this disease locomotion and vocalization are greatly interfered with, and sometimes abolished, while there are at the same time failure of the heart's action and weak inspiratory power. In protracted and uncomplicated cases, it may be shown during life that the chest's capacity is diminished, that the movements of the thoracic walls are restricted, and that the respiratory murmurs are feeble; and it is not to be wondered at, therefore, that some diminution of volume and change of consistence are found in the lungs after death. These organs are of pale colour, of dry texture, and less firm and resisting than they ought to be, having lost their elastic resilience, so that they are capable of being squeezed into a small compass. The air vesicles are of large size and the bronchi look as if thinned. The process upon which these changes depend is, in all probability, essentially one of withering and obliteration of capillaries dependent on diminished respiratory function and blood volume.

The other pathological changes encountered in cases of general paralysis, may be best considered as they affected the pleural covering of the lungs or the lung substance, as congestion, consolidation, or phthisical degeneration.

Changes in the Pleuræ.—The pleural sac enclosing the lung is more given to morbid affections than any other serous membrane, and in persons labouring under general paralysis it seems to be even more prone to abnormal conditions than amongst mankind generally. Such abnormal conditions, which were almost invariably the result of inflammation, were found in 64 of the 100 cases of general paralysis which I have made the subject of special examination as regards the state of the lungs. The nature and distribution of these abnormal conditions are indicated in Table III.

From this Table it appears that in 36 general paralytics out of 100, the pleuræ were in a normal state and free from adhesions, but that in the remaining 74 cases pathological changes of one kind or another were noted. In 5 cases there

were unmistakable signs of recent inflammation of one or both pleuræ, and in 71 cases there were changes in the pleural sacs which might be fairly regarded as products of past inflammatory attacks. In those cases in which the adhesions were few in number and limited in extent, it might be said that they were not necessarily the result of inflammation properly so-called, but of the hyperplastic and proliferative processes which are so common in serous membranes, and which leave behind them pleural adhesions in so large a proportion of persons who have passed middle life; but even in those cases included in this Table, along with the whitish bands or patches of adventitious membrane connecting the pleuræ, there were in many

TABLE III.

SHOWING STATE OF THE PLEURÆ IN ONE HUNDRED CASES OF GENERAL PARALYSIS.

State of Pleuræ.	Males.	Females.	Total.
Pleuræ on both sides, free from adhesions and normal	30	6	36
Old, slight, and scattered adhesions on both sides	13	5	18
Old, strong, and general adhesions on both sides	8	4	12
Old adhesions confined to right side	19	4	23
Old adhesions confined to left side	5	1	6
Recent adhesions on both sides	1	-	1
Recent adhesions on right side	1	-	1
Recent adhesions on left side	1	-	1
Fluid in the pleural cavities	2	-	2
Total	80	20	100

instances changes in the pulmonary substance beneath, which pointed to their origin in inflammatory lymph. And in the much greater number of cases, in which the adhesions were wide-spread, or general and firmly organized, the conclusion was irresistible that they had arisen out of pleurisies, which had often perhaps attracted little or no attention. Of course, in a large proportion of these cases, a proportion which, however, it was impossible to estimate, the pleurisy had occurred before the incursion of the general paralysis; but still the facts, that traces of 5 recent pleurisies were found in 100 subjects, and that many of the adhesions which are tabulated as old were only comparatively so, and had probably been formed within two

years of the time of death, justify the belief that there is a special proclivity in general paralysis to inflammation of the pleural membrane. As will be seen presently, the sufferers from general paralysis are particularly liable to attacks of pulmonary congestion, which may readily spread to the pleuræ. They are in the prime of life when pleural inflammations are most frequent, and they are apt heedlessly to expose themselves to changes of temperature and currents of cold air. Anstie thought that extreme muscular over-exertion and strain, as in public speaking, sometimes produces pleurisy in otherwise healthy persons, and if this supposition is correct, it is easy to understand why pleurisy should be common amongst general paralytics, who, when excited, in the earlier stages of their malady, will talk or shout incessantly for hours, and even for days and nights together, with an energy and persistency that the most inveterate orator never attained to. And perhaps another explanation of the frequency of pleurisy in general paralytics may yet be discovered in some constitutional tendency to which both of these diseases are etiologically related.

When the adhesions between the pleura were old they were composed of tough white connective tissues; when they were recent they were formed of thick layers of yellowish lymph; and where effusion had taken place, the fluid was turbid and contained purulent matter and flakes of lymph.

It will be observed that where the pleuritic adhesions were confined to one side, they were located on the right side more frequently than on the left, in the proportions of four to one.

Congestion of the Lungs.—Of all pathological changes in the lungs in general paralysis, congestion is the most common, and this is often present to a degree much greater than one is accustomed to find after slow death from exhausting bodily diseases. It was present to a marked extent in 49 out of the 100 cases of general paralysis which are here dealt with, and in many of these its intensity was such that it was difficult to say that it did not amount to pneumonic consolidation. In 38 of the 49 cases, it existed in both lungs, in 11 cases it was limited to the right lung and in 6 to the left, and whether involving one

TABLE IV.

SHOWING THE WEIGHT OF THE LUNGS IN THIRTY-TWO CASES OF GENERAL PARALYSIS IN WHICH THESE ORGANS WERE BOTH CONGESTED.

——		Right Lung.						Left Lung.					
		Highest.		Lowest.		Mean.		Highest.		Lowest.		Mean.	
		Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.
Persons .	32	35·00	992	12·00	340	24·50	698	31·75	903	14·00	395	22·50	641
Males .	24	35·00	992	17·75	505	27·25	773	31·75	903	16·25	460	25·00	709
Females .	8	21·25	602	12·00	340	17·75	505	18·75	530	14·00	395	15·25	435

or both lungs it was invariably more extreme, and extensively diffused in the male than in the female sex. In Table IV. is set forth the weight of the lungs in those cases in which both were congested, and here we see that the vascular engorgement and œdema caused an increase of weight of 5 oz. in the right, and of 5½ oz. in the left lung in the male, but of only a quarter of an ounce in each lung in the female. This observation is interesting in connection with the well-known fact, that women die of pneumonia in much smaller proportion than men do, and that that disease is, as a rule, much milder in them than in the other sex.

The congestion of the lungs in general paralysis is of course hypostatic in character, and is almost invariably most marked posteriorly, and in the lower lobe. But it is by no means limited to this pulmonary region, for in 25 of the 49 cases in which it existed, it is described as having extended throughout the whole lung. In 22 cases it was confined to the lower lobe, and in 2 cases (in one of which it was associated with an abscess, and in the other with miliary tubercles) it involved the upper lobe alone.

The amount of congestion varied from slight hyperæmia, with some serous infiltration of the pulmonary parenchyma, up to extensive engorgement of a dark red, or even blackish colour. The boundary line separating the cases of most pronounced congestion from those of actual pneumonia was certainly not well defined, and some of the cases tabulated as congestion might have been perhaps not improperly called pneumonia. The presence of even obscure crepitation in the congested lung tissue, the escape of frothy fluid from it on section, and the restoration in some degree of the natural colour of the lung by prolonged washing, were taken as signs that congestion had not actually passed over into the pneumonic condition. With the congestion there was constantly bronchial catarrh. In 17 cases pus was found in the bronchi, and in 30 cases there was frothy mucus with increased vascularity of their lining membrane.

The hypostatic congestion of general paralysis is not difficult to account for. The sufferers from that protracted and debilitating disease have their muscular strength reduced to a

low ebb, and in them respiration is carried on, as we have seen, with little vigour. Incapable in many instances of voluntarily changing their position, they remain for prolonged periods in attitudes favouring the accumulation of blood in dependent parts, while a loss of power in the nervous mechanism, regulating the calibre of the vessels, is also conducive to local congestions. It is possible that in the advanced stages of general paralysis, there may be some degeneration of the excito-motor ganglionic elements, which exist in the plexuses surrounding the pulmonary vessels, in the inferior thoracic or superior cervical ganglion, in the communicating nerves, in the antero-lateral columns of the cord or in the vaso-motor centre in the medulla oblongata. But it is not necessary to suppose changes in any of these in order to explain the vaso-motor disturbances, which are evident at an early period in the course of general paralysis, and which some have regarded as an essential element in the disease, for recent experiments have demonstrated, that the vaso-motor centre in the medulla is linked with certain portions of the cerebral cortex. Budge has proved that irritation of the crus-cerebri induces arterial contraction, and Eulenburg and Landois have shown that definite areas of the cerebral hemispheres in dogs, corresponding to the central convolutions in man, constitute vaso-motor centres for the extremities of the opposite half of the body. These vaso-motor centres, destruction of which by the cautery leads to a more or less enduring increase of temperature in the limbs of the opposite side, are situated in the vicinity of the voluntary motor centres of the cortex, which are invariably involved in the degeneration of general paralysis. And if vaso-motor centres for the limbs are located there, it may be legitimately inferred that vaso-motor centres for the head and neck, thorax and viscera are not far off, in the irritation or destruction of which ample explanation is to be found of the pallors, flushings and perspirations, the local changes of temperature and the internal congestions which are so frequently noticed in general paralysis.

A practical lesson may be derived from the great liability of those who labour under general paralysis, to suffer from pulmonary congestion, and it is to this effect, that their days

may be prolonged by careful attention to their decubitus. A mere change of posture may remove an hypostatic congestion which would soon deepen to such an extent as to curtail life, were the position of the body left unchanged, and careful nursing, by night and day, may do much to ward off complications of this kind. General paralytics who are reduced to helplessness should be systematically moved from side to side, when in bed, and from one attitude to another when occupying a sofa or chair.

It seems to me also, that the liability of general paralytics to pulmonary hypostasis affords a strong argument against that re-introduction of mechanical restraint into our asylums, which finds some advocates in these days. Were mechanical restraint sanctioned, it would probably be had recourse to more frequently in the case of general paralytics than in that of any other class of lunatics. The reckless violence and destructiveness which so often occur at the outset of the disease, and the tottering restlessness of its closing scenes, with the risk of fractured ribs which attends its whole course, would all recommend the employment of what is, at any rate, a sure method of avoiding some distressing accidents. But it is just in general paralysis that special dangers would wait on this sure method. In the stage of excitement there is the danger of pulmonary fluxion, and in the stage of dementia that of pulmonary stagnation. Bonds restraining the muscles are very apt to induce pulmonary fluxion where there are nervous erethism and palpitation, and Niemeyer speaks of scandalous cases of frequent occurrence, in which delirious persons, or patients with delirium tremens, having been brutally strapped to their beds, are found dead the next morning with bloody foam upon their lips Then, appliances preventing free movement may cause, it is scarcely necessary to point out, passive congestion and œdema of the lungs, while the trouble of removing and reapplying restraint would inevitably interfere with those frequent changes of posture which are so desirable in advanced cases of general paralysis.

Pneumonia.—Distinctly pneumonic conditions of the lungs were found in 13 out of the 100 cases of general paralysis, the post-mortem reports of which we have been examining.

Two of the 13 cases might be regarded as simple or ordinary acute pneumonia with profuse fibrinous exudation, and the remaining 11 as instances of broncho-pneumonia and hypostatic pneumonia. Both lungs were involved in the inflammation in 8 cases, the right lung alone in 4 cases, and the left lung alone in 1 case. Only in 1 instance was a pneumonia found in the body of a female; 12 of the patients who laboured under this disease at the time of death being males. The increase in the weight of the lungs caused by the pneumonic process was very remarkable. In one male, in whom both lungs were in a state of gray hepatization, the right one weighed $46\frac{3}{4}$ oz., or 1326 grammes, and the left $49\frac{3}{4}$ oz., or 1407 grammes. In another male, in whom the left lung was in a state of red hepatization, that organ weighed $56\frac{1}{2}$ oz., or 1600 grammes, while the sound lung of the other side weighed $23\frac{3}{4}$ oz., or 670 grammes. The mean weights of the lungs in the 8 cases (all males), in which both lungs were inflamed, was $35\frac{1}{2}$ oz., or 100 grammes, for the right, and 33 oz., or 936 grammes, for the left lung.

As has been pointed out, in these cases of pneumonia, both lungs were inflamed in a large majority of instances, and in this respect the pneumonia of general paralysis resembles the congestive pneumonia described by Mr. Erichsen as the commonest cause of a fatal issue after capital operations, when of course, debility, prolonged recumbency, and nervous shock, combine to favour its occurrence. In Mr. Erichsen's cases the right lung alone was affected three times, the left lung alone also three times, and both lungs together, twenty-two times. But in ordinary sthenic pneumonia the right lung is most frequently affected, the left lung next, and lastly, both lungs conjointly. Thus Huss found that in 53 per cent. of the 2,616 cases included in his statistics the pneumonia was on the right side, while it was on the left side in 32 per cent., and on both sides in 15 per cent. Grisolle states that in 1,430 cases of pneumonia, which he collected from various authors, the inflammation was in the right lung 742 times, in the left lung 426 times, and in both lungs 262 times. But while, contrary to the ordinary rule in the pneumonia of general paralysis, as well as in the pulmonary

congestion of that disease, both lungs are involved much oftener than either lung singly, it is to be borne in mind that when one lung alone is affected by inflammation or congestion, it is the right lung that most frequently suffers. Rosenbach has shown[1] that in acute diseases of the respiratory organs in cases of hemiplegia, the lung of the paralysed side is much oftener the seat of disease than that of the sound side. In trying to account for this, he discovered that the direct reflex irritability of the involuntary muscular tissue of the paralysed side is decidedly lowered, one result of which is that foreign matter, such as saliva, entering the bronchi of the paralysed side does not give rise to sufficient expulsive efforts, so that unilateral broncho-pneumonia is set up. Founding on Rosenbach's observations, it might perhaps be argued that, as in general paralysis the left or leading hemisphere of the brain suffers probably earlier and more profoundly than does the right hemisphere, and as lesions of the cortex always cause functional and organic derangements on the opposite side of the body, so the right lung must inevitably suffer oftener than the left one. But this theory affords no clue to the preference of pneumonia for the right lung in persons in whom there are no lesions of the cortex, and some more general explanation is yet needed of the right side tendencies not only of pneumonia and congestion of the lungs, but also of pleurisy.

When seeking for an explanation of the great prevalence of pneumonia in general paralytics, the classical researches of Traube at once come to mind. Traube demonstrated that section of the vagi in the neck in animals, produces inflammation of the lungs and early death; and he referred the pneumonia which so frequently follows the operation to paralysis of the digestive canal from the throat to the stomach, and the consequent accumulation in the œsophagus, and overflow or aspiration into the air passages, of the secretions of the mouth and particles of food. Steiner subsequently showed that ligature of the œsophagus at its middle point, with section of the recurrent laryngeal nerves, produced exactly the same results which Traube obtained on section of the vagi; and then, dividing the vagi longitudinally into two bundles, which he was

[1] 'Centralblatt,' 1879, No. 16.

able to differentiate as motor and sensory, he further showed that section of the motor bundle was followed by an extensive pneumonia in a few days, due, like the pneumonia following complete section of the vagi, to paralysis of the œsophagus and larynx; and that, while section of the sensory bundles alone did not induce pneumonia, but only irregularity of respiration, their section immediately subsequent to section of the motor bundles played some part in the succeeding inflammation, by leading, through the powerful aspiratory efforts induced, to the carrying into the bronchi of more fluid than would otherwise have reached them. Now, in almost all general paralytics in the last stage of the disease, there is more or less paralysis of the œsophagus and larynx, with accumulation of food and fluid in the pharynx, and imperfect closure of the glottis. Choking is a common occurrence amongst them, and has to be specially provided for, and it might seem, therefore, that the mechanical or non-trophic theory of the causation of broncho-pneumonia was fully applicable in their case. The objection to this theory that particles of food are rarely or never found in the bronchi of the inflamed lungs is disposed of by the observations of Frey* that the mere trickling or injection of saliva into the smaller bronchi is sufficient to set up inflammation. But while fully admitting that mere mechanical irritation from an *error loci* of the ingesta or secretions may play an important part in the causation of pneumonia in general paralysis, it must, I think, be maintained that there are other factors in its etiology which must not be lost sight of. Solutions of nitrate of silver have been injected into the bronchi of persons free from nervous disease, without setting up pneumonia, and amongst general paralytics cases of broncho-pneumonia occasionally happen which can be distinctly traced to a reflex cause, such as exposure to a cold draught. It is probably with the pneumonia of general paralysis very much as it is with the bedsores in that disease, of which the etiology is complex. Mechanical irritation by frequent wetting and prolonged pressure, is highly instrumental in inducing these bedsores, and great care in avoiding mechanical irritation may effectually prevent their occurrence. In the Royal Naval

* 'Lo Sperimentale,' Jan. 1878.

Hospital at Yarmouth, where a large number of severe cases of general paralysis are always under treatment, a bedsore is never seen, and the officers pride themselves on their skill in preventing their occurrence. But on the other hand, no one who has studied the bedsores which occur in such cases, will doubt that they are produced by a degree of mechanical irritation which would have no such effect in a healthy person, and that they are sometimes developed with a rapidity which betokens a profound disturbance of innervation in the part, as does also their tendency to dangerous extension. And so in the pneumonias, as in the acute and chronic decubitus of general paralysis we are forced to assume that besides mechanical irritation, some centric nervous influence is at work, causing neuro-paralytic hyperæmia, and perhaps a suspension of healthy trophic influences, for the existence of trophic fibres in the lungs is not yet finally disproved.

Phthisis.—Phthisical consolidation, or disintegration of lung tissue, was observed in 25 out of the 100 cases of general paralysis analysed, and in the subjoined Table, some information as to the weight of the lungs in these cases is supplied. The effect of phthisical disease is obviously very materially to augment the weight of the lungs which it invades. The mean weight of the lungs included in Table V. is not only much above the mean weight of the normal lungs in general paralytics, but considerably exceeds the mean weight of the lungs when congested. The mean weight of the right lung in the phthisical cases, was 6 oz., or 166 grammes, in the male, and $2\frac{1}{2}$ oz., or 66 grammes in the female—greater than the mean weight of the right lung in the normal cases, and 1 oz., or 27 grammes, in the male, and $2\frac{1}{4}$ oz., or 60 grammes, in the female, greater than the mean weight of the lungs in the cases in which there was congestion. The mean weight of the left lung in the phthisical cases was $5\frac{3}{4}$ oz., or 167 grammes, in the male, greater than the normal standard, but in the female the mean weight of this lung was a trifle short of that standard. As compared with the cases in which there was congestion, the mean weight of the lungs in the phthisical cases was slightly in excess in the male, and slightly deficient in the female.

TABLE V.

SHOWING THE WEIGHT OF THE LUNGS IN TWENTY-FIVE PERSONS LABOURING UNDER GENERAL PARALYSIS AND PHTHISICAL DISEASE.

——		Right Lung.						Left Lung.					
		Highest.		Lowest.		Mean.		Highest.		Lowest.		Mean.	
		Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.
Persons .	25	40·75	1155	7·50	214	25·50	725	32·00	905	10·50	298	22·00	621
Males .	17	40·75	1155	20·75	590	28·25	800	32·00	905	15·00	425	25·25	718
Females .	8	27·00	765	7·50	214	20·00	565	20·50	578	10·50	298	14·75	417

In six of the cases included in Table V., only the remnants of past phthisical disease were found in the lungs. These consisted of nodules of earthy or chalky matter, situated at the apex of the lung in every case, and occupying one apex in four, and both apices in two cases. Around these nodules or masses, which were single in four cases and double in one, and which varied in dimensions from the size of a pea to that of a walnut, the lung tissue was invariably dark and indurated, while over them the pulmonary surface was more or less puckered. As it is highly improbable that resolution with absorption of intra-alveolar exudation, and drying up of tuberculous deposits, could take place during the progress of so debilitating a malady as general paralysis, it may be fairly inferred that in these cases the phthisical affection had existed, and had been practically cured long before the supervention of the cerebral disease. In the remaining cases, however, in which phthisical changes were found in the lungs, these were of a kind indicating their recent origin and active state. They were confined to the right lung in 3 cases (all males), to the left lung in 7 cases (4 males, and 3 females), and involved both lungs in 9 cases (5 males and 4 females). They consisted in grey miliary tubercles in 9 cases, in caseous masses in 5 cases, and in mixed grey granulations, and yellow cheesy masses in 5 cases. Cavities were associated with the miliary tubercles in 3 cases, and with the mixed tubercular and caseous degeneration in 4 cases, being in 5 cases confined to the upper lobe, and in 1 to the lower lobe. The vomicæ were always of comparatively small size, with ragged walls, presenting no appearances of a false membrane, and the lung tissue around the miliary tubercles and nodules was for the most part congested and œdematous, and but little indurated. There was no room for doubt that in all these 19 cases, the inflammatory processes leading to phthisical consolidation, and destruction of lung tissue, had arisen during the course of the general paralysis, and had been cut short by the natural termination of that disease. In none of them had the disorganisation spread to that extent which we are accustomed to see in the lungs of patients who have died of phthisis.

Now the age at which patients who perish by general

paralysis are attacked by it, and their previous history and physical characteristics, alike forbid us to attribute any phthisical disease which may be developed in them to the influence of hereditary predisposition. They have passed the time of life at which hereditary phthisis most frequently displays itself, and they have never exhibited that constitutional feebleness which is so characteristic of those who have an inherited tendency to that disease. They are as a rule vigorous and much enduring, until general paralysis lays its grasp upon them; we must therefore look for the personal factor in the causation of phthisis, when they suffer from it, not in an inherited, but in an acquired state of health, unless, indeed, certain neurotic tendencies which the sufferers from general paralysis and phthisis both exhibit, be regarded as having an etiological relation to the latter disease. And this acquired state of health is readily recognised in the general debility and impairment of power which constantly accompany general paralysis. Here we have that condition of the system which favours the development and progress of phthisis, when inflammatory changes are set up by any irritation or injury of the pulmonary tissue. And such irritation or injury is of course peculiarly likely to occur in the victims of general paralysis, whose mental disquietude and muscular helplessness, are alike apt to lead to chilling of the body by exposure to cold, and who are, as we have seen, exceedingly susceptible to pulmonary congestion and bronchial catarrh.

But there is another respect in which general paralytics may be regarded as favourably situated for contracting phthisis. They are generally, during their latter days at any rate, in close association with persons suffering from this disease, and they inhabit buildings in which it has been long established. Dr. Villemin, when speaking of the extraordinary prevalence of phthisis amongst soldiers, expressed his conviction that the barrack is to the soldier, in the production of consumption, what the regimental stable is to the horse in the development of farcy; and evidence is now accumulating daily in support of the transmissibility of phthisis from one person to another.

The theory of Koch, that the tubercle bacillus is essential to the development of the phthisical process, and that

phthisis is therefore a communicable disease, although not fully established, is daily becoming more probable, and the history of the connection of phthisis with general paralysis seems to lend it some confirmation. The prevalence of phthisical destruction of lung tissue amongst general paralytics is very remarkable. In my cases it existed in 19 per cent. But the prevalence of phthisis, not merely amongst general paralytics, but amongst the insane generally, is noteworthy. While in the population of England, above twenty years of age, phthisis is responsible for 14 per cent. of the whole mortality, in the public lunatic asylums of England and Wales, the population of which may be taken as above twenty years of age, phthisis is the assigned cause of death in 15 per cent. of the whole mortality; while it exists at the time of death in a considerable proportion of cases of general paralysis, epilepsy, &c., in which it is not the certified cause of death, and so does not appear in the tables of mortality.

Now a certain proportion of patients, when admitted into lunatic asylums, are labouring under phthisis, but, on the other hand, a certain proportion of patients are discharged from these establishments when labouring under that disease; and it is, I believe, incontestable that phthisis is generated in lunatic hospitals to a greater extent than in the general community. Bearing in mind that the inmates of asylums are well fed, clothed, and housed, and live in situations specially selected for their salubriousness, and under strict hygienic regulations, the prevalence of phthisis amongst them strongly suggests the propagation of the disease by contagion. And this suggestion has additional probability given to it by several other facts and considerations. While in the general community the mortality of males from phthisis is greater than that of females, in lunatic asylums the reverse is the case. In England and Wales, phthisis is the assigned cause of death in 15·2 per cent. of the whole mortality amongst males, and in 13·4 per cent. of the whole mortality amongst females; but in the pauper lunatic asylums of England and Wales it is the assigned cause of death in 12 per cent. of the whole mortality amongst males, and in 17 per cent. of the whole mortality amongst females. Now, the chief respects in which female lunatics differ from

male lunatics in their mode of life, are that they are more sedentary in their habits, and are less out of doors, being more confined to their day-rooms and wards, and compelled to breathe more constantly the air which is breathed, and possibly poisoned, by the patients who are consumptive. And further than this, it might be shown that the mortality from phthisis has been materially diminished in some asylums by increased attention to sanitation, the ventilation of the buildings, and the out-door exercise of the patients, by the adoption, in fact, of means calculated to lessen the risk of dissemination of contagious or infective matter in the atmosphere. It may also be shown, that in every asylum phthisis originates most frequently in patients inhabiting wards such as the infirmary wards, in which the air is liable to be contaminated by the breath and sputum of phthisical patients. And thus it is, I should be inclined to maintain, that phthisical conditions are so common in general paralytics. They are, for the most part, from the first, unfit for out-door employment, and in the latter stage of their illness they are, almost without exception, relegated to the infirmary wards, so that they may have the benefit of better nursing. But in these wards they are very generally in association with patients sinking under phthisis; and the fact that 19 per cent. of general paralytics dying in asylums present evidences of recent phthisical disease, whereas phthisis is responsible for only 15 per cent. of lunatic asylum mortality as a whole, seems to me to support the view that phthisis is communicable, and is dependent on organisms which are specific. That the tubercle bacillus is a constant attendant on tubercular processes cannot now be denied, that it is the true cause of their infective property is most likely, and that it may be communicated from man to man, and set up phthisical changes anew whenever it lights on a congenial soil, is rendered probable by facts such as those which have been adduced regarding the connection of phthisis with general paralysis.

Until Koch's theory is disproved, it would be prudent to act on the assumption that it is true, and to prevent the close association of persons actually suffering from phthisis with those who, from inherited tendency or deterioration of health, are especially liable to contract the disease. A large number

of lunatic asylums have now detached hospitals for contagious diseases, which fortunately stand empty for a great part of the year, and it might be well to isolate in those buildings all cases of phthisis. The experiment could do no possible harm, and there is every prospect that it would be attended with benefit to the victims of phthisis, and with safety to those who are in danger of its attacks.

Abscess of the Lung.—In 2 cases out of the 100, abscesses of the lung were discovered. In one of these the abscess, which was of the size of a small orange, and situated in the upper lobe of the right lung, was clearly the consequence of a pneumonia, but in the other, the abscesses (for there were three of small size) were probably of metastatic origin. They were in the lower lobe of the right lung of a man, in whom extensive bedsores had formed almost suddenly on the sacro-gluteal region a few weeks previously, and who had suffered from remittent fever. There was no tubercular matter in either lung in this man, and each abscess had surrounding it a ring of consolidated pulmonary tissue.

Gangrene of the Lung.—Dr. Mickle has seen gangrene of the lung in general paralysis, but nothing approaching it was observed in any of the 100 cases which I have been analysing.

SPONTANEOUS POSTURES OF THE HAND CONSIDERED AS INDICATIONS OF THE CONDITION OF THE BRAIN.[1]

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THE term posture is here used as it is commonly understood, to indicate the relative position of the several members of the body with regard to one another, or the relative position of the individual parts of a member.

It is proposed to speak in this paper of only a few spontaneous postures of the hand.

The postures of a limb depend in their immediate mechanism upon the resultant action of opposing muscles, the relative tone of the antagonistic flexors and extensors, the adductors and abductors, etc. Various views may be held with regard to the nerve-mechanism which regulates the contraction of opposing sets of muscles. That mechanism may be considered a line of reflex action regulating the action of the muscles, or the balance may be considered due to some other kind of nerve-mechanism. In any case it will, I think, be granted that some portion of the central nerve-system is the cause of that balance of the muscles that produces the posture, and therefore the posture is an index of the condition of that central nerve-mechanism.

In examining the condition of the nervous system in children, it has for several years been my habit to observe what spontaneous posture would be assumed by the hand

[1] Read before the Royal Medical and Chirurgical Society of London Nov. 28th, 1882.

when the forearm was held out.[1] Let a weak, nervous child be requested to hold out her hands in front on a level with the shoulder in the prone position. The limb is now free or disengaged, and the posture assumed by the hand is in most cases, I believe, the spontaneous outcome of the action of the nerve-centres.

Let me describe the spontaneous posture of the hand usually seen in a nervous child, and then discuss the causation and meaning of this posture.

In this posture—"the nervous hand"—the wrist is slightly flexed, or bent, the metacarpo-phalangeal joints are moderately hyper-extended (extended beyond the straight line), the first and second internodes being either slightly flexed or kept straight. The thumb is extended backwards, and somewhat abducted from the fingers. This spontaneous posture I have seen, and others with me, in hundreds of cases, usually in nervous children, bad sleepers, those convalescent from chorea, etc.; the same spontaneous posture is sometimes seen in partial hemiplegia. The posture is often bilateral, but is usually unequally represented on the two sides, it is also often seen on one side only, especially in children convalescent from hemi-chorea. From continued observation of spontaneous postures, and after frequently trying what spontaneous posture would be assumed by the free hand in various subjects, cases of weakness, strong men, feeble women, nervous children, an empirical idea was obtained as to the indication of each posture. Then comparison, analysis and analogy enabled me to suggest some definite principles. In seeking a rational explanation of this posture, I first tried to find a solution by looking at it after the manner in which Charles Darwin made most of his observations upon postures. Cases were looked for where this posture of the hand was assumed or brought about for some useful purpose, or its occurrence was attended with some kind of amelioration of weakness. Then this "nervous hand" was seen (in a hand not free or disengaged) in people who, standing, lean a little forward with the outspread hands resting on the table for support. This hand seeking rest droops at the wrist, and then if the stimulus to the muscles is

[1] See 'Brain,' Part XI.; and 'British Medical Journal,' Dec. 6, 1879.

weak, as the weight of the trunk bears upon the resting hands, hyper-extension of the metacarpo-phalangeal joints, and of the thumb, is mechanically brought about. If this be accepted as an explanation, the argument stands thus:—

1. Observation shows that when a man is tired or weak he often rests his hands as described.

2. The nerve-mechanism corresponding to the hand is thrown into the special condition corresponding to the "nervous hand," and thus that posture is mechanically brought about.

3. The nerve-mechanism corresponding to the hand falls spontaneously into the condition corresponding to the "nervous hand" when the man is tired, or weak.

4. When the hand of a weak man is held out free, its nerve-mechanism spontaneously places it in the "nervous posture."

The attempt at explanation may be made again, commencing with the principle of analysis and then applying the principle of analogy.

Anatomical analysis shows that this "nervous hand" presents two principal elementary conditions:

(1) Flexion or drooping of the wrist.

(2) Hyper-extension of the metacarpo-phalangeal (small) joints.

So much for analysis, now as to analogy.

Looking for analogy between postures, flexion, or drooping of the wrist, may be seen as in a hand that is resting, in a hand that is passively held out while the patient is deeply asleep, or in deep coma, or in a paralysed arm. It is probable then that the wrist flexion indicates the weakness of the "nervous hand."

Hyper-extension of the metacarpo-phalangeal joints and thumb may be seen as a temporary condition in subsultus tendinum, and chorea. In all these cases the unstable condition of the nerve-mechanism is indicated by the extensor movements of small parts.

Now looking at the elementary conditions shown by analysis to constitute the "nervous hand," and the probable indication of the conditions analogous to these, we get some kind of explanation of this posture. The drooping wrist is analogous to that seen in hemiplegia; the hyper-extension of the knuckles

and thumb is analogous to conditions such as chorea, and subsultus tendinum. This appears a more rational explanation than that first given, and the methods of analysis and analogy give some indication of the physiological significance of the posture. Something very like this posture of the hand is indicated by Dr. Little in his work on deformities, 1853, as the result of "spastic contraction."

Another posture of the hand less frequently seen in pathological states, I described under the name of the "Energetic hand," and since so doing I have found the posture figured by H. Meillet of Paris, with the note "Main dite du prédicateur emphatique. La Salpêtrière, service de M. Charcot, salle Saint-Paul, No. 6, Isméric Angot." It is there figured as a permanent deformity resulting from brain disease. Dr. Little also figures this posture as due to spastic contraction. In this posture the wrist is extended, and the small joints are all in flexion. Here analysis shows the large joint, the wrist, in extension, the opposite to the condition of weakness, and this extension of the large joint gives the posture the indication of excitement in the nerve-mechanism. The small joints are all flexed, as seen in the hand of a man in sleep or resting, and this gives the posture the indication of strength or activity with rest.

Anatomical analysis shows the "energetic hand" to be the antithesis of the "nervous-hand."

	Nervous Hand.	Energetic Hand.
Wrist	flexed	extended
Metacarpo-phalangeals	extended	flexed
1st and 2nd internodes	flexed	flexed
Thumb; metacarpo-phalangeal	extended	flexed
Thumb; internode	extended	flexed
Phalanges; relative position	slightly abducted.	adducted

The physiological expressions of the two postures are also opposites.

This suggests another principle of use in explaining the meaning of certain postures. The principle of antithesis[1] may be enunciated as follows:—"In opposite conditions of the

[1] Charles Darwin uses the principle of Antithesis to explain many modes of Expression. See 'Expression of the Emotions,' p. 28.

nervous mechanisms producing the postures of a joint, the postures will be opposite or antithetical"; e.g., in a weak or inactive condition of the nerve-mechanism governing the wrist flexion results; this would lead us to anticipate that in an excited condition of this piece of nerve-mechanism we should find the wrist extended.

My present object is to show that spontaneous postures may be studied with advantage as indications of the conditions of the brain. As to the principles involved in these inquiries. A primary postulate, involved in the following principles, is that—

"If we see some spontaneous nerve-muscular action often repeated in the same and in different subjects, it may be assumed that there is some nerve-centre, or nerve-mechanism, corresponding which can act independently." In any particular instance the assumption of the existence of such a centre or piece of nerve-mechanism would be strengthened if it could be shown that there are cases of excitation and cases of paralysis of such hypothetical centre.

In analysing any posture of the hand we may consider the bearing of the following Principles, or points of analysis, upon the subject:—

I. Describe the posture indicating the anatomical condition of each joint.

II. Consider the posture of the small parts of the limb as distinguished from the larger parts.

In many other fields of observation the importance of this rule has been illustrated thus:—the small parts of a member are the most affected in chorea, in hemiplegia, in subsultus tendinum.

III. Consider the different relative postures seen in the large and small joints.

Interdifferentiation of the postures of the large and small joints may give important indications: the large joint may be paretic while the small joints are in extension, from irritability of the nerve-centres which govern their movements. In such a case there would be an interdifferentiation of the nerve-centres governing the different joints.

IV. Consider the relative condition of posture in collateral

joints. The knuckles are collateral joints, the posture of each may be different; so there may be differences in the posture of the collateral first inter-phalangeal joints of the fingers. Collateral interdifferentiation is usually seen in the movements of the hand in chorea, but sometimes all the fingers open and close together. Collateral differentiation is marked in athetosis.

V. Consider if the posture is symmetrical.

A posture may be called symmetrical when the parts of the member are equally placed or balanced about the axis of the limb. Symmetry therefore indicates the exact balance of opposing muscles, and consequently the exact balance of the portions of the nerve-mechanism which cause the postures of the limbs.

VI. Consider if there be any indication of excitement or over-action of any nerve-centre or centres usually weak, such as the centre or nerve-mechanism governing the extensor movement of a joint.

It is certain that extension of a joint is usually a movement of less power or strength than flexion of the joint. Observations, comparisons and analysis seem to indicate the nerve-centre, or mechanism governing the extension of a joint (i.e. the action of the weaker muscles) as being weaker than the centre governing flexion. Observe if such indications of excitement subside in sleep, they may do so in chorea; some cases lose the "nervous hand" during sleep, the hand when held out free falling into the posture of the "hand in rest."

VII. If there be general, equal, widespread stimulation or weakness of portions of the nerve-mechanism governing the postures of a limb, or part of the body, the action of the stronger muscles prevails. General weakness may be indicated by general preponderance of weak centres as in general extension. General weakness may also be indicated by general slight flexion, showing all the nerve-mechanism paretic and the stronger flexor muscles prevailing.

VIII. Consider in the posture if any joint or set of joints be in position analogous to that of any other posture, and whether the significance of the position of that joint be the same as in the posture to which the analogy is made.

IX. Consider if the posture be the antithesis of any other known spontaneous posture.

It is probable that in many cases opposite conditions of the nerve-mechanism producing postures will produce antithetical postures.

The following suggestions are offered as to the *methods of determining whether a posture is the outcome of the spontaneous action of the nerve-centres.*

A posture observed can only be considered as the outcome of the spontaneous action of the nerve-mechanism when the limb or part is free and disengaged. If the muscles of the hand are engaged in holding an object, or in an act of manipulation, the postures of the hand are not simply the outcome of the spontaneous action of the nerve-mechanism.

Postures are frequently seen accompanying, and apparently caused by, demonstrable brain-disease; as examples the "convulsive hand," cerebral facial palsy. When these postures occur otherwise, apparently spontaneously, they may be looked upon as nerve-muscular signs produced by the nerve-mechanism corresponding.

Postures which are frequently seen in very young children and infants are certainly spontaneous. Postures seen to be frequently repeated in many such young subjects are probably spontaneous nerve-muscular actions. With regard to postures thus observed in many infants it is probable that the piece of nerve-mechanism corresponding is well developed, and rendered very definite so as to be easily excited to spontaneous action.

When a posture seen in an adult is found to be analogous to one often seen in infants, the probability of its spontaneous origin is strengthened.

Postures of disengaged parts, still observed in man, and frequently represented in ancient art, are probably of frequent spontaneous occurrence, and highly characteristic nerve-muscular postures.

The application of the "principles of analysis" may assist in determining whether a certain posture observed is probably the outcome of the spontaneous action of the nerve-centres.

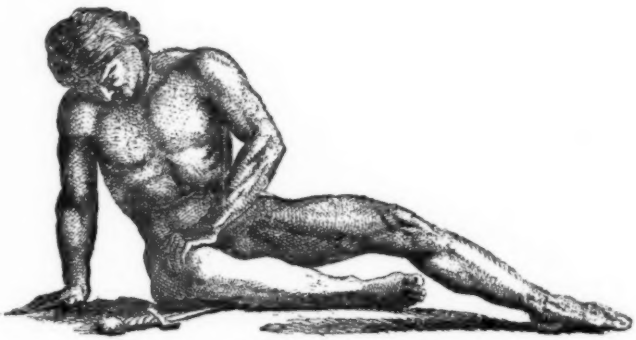
In these studies care must be taken to avoid certain errors

from attributing all postures to the action of the central nerve-mechanism.

Postures may be due to joint-disease, especially Arthritis deformans. M. Charcot carefully differentiates between this deformity, and the posture he has described as the "writing hand" often seen in Paralysis agitans.

Postures may be due to local causes—tumours, local inflammation, etc.

Postures may be determined by organic conditions such as difficulties in the respiratory and circulatory organs causing orthopnœa. Sir. C. Bell drew attention to this matter in his critical analysis of the posture of the Dying Gladiator. Such postures are not the direct effect of the spontaneous action of the nerve-centres.



THE DYING GLADIATOR.
(*After a drawing by Sir C. Bell.*)

Gravity may be a factor in the causation of postures, whether the member be "free" or "engaged," thus: In the figure of Hercules at rest, he leans on a vertical club to support his body, and the posture of the right arm is determined mainly by gravity. In a paretic arm such as is often seen in chorea, or from brain disease, the wrist will droop into flexion if the forearm is held out prone, but will fall into extension if the forearm is held out supine. Dorsal decubitus results from gravity, together with general palsy of the motor nerve-mechanism.

Postures may be due to local nerve injury or disease, thus: Injury to the facial nerve; injury to the musculo-spiral nerve; a gumma pressing upon the third cranial nerve, may produce certain postures of the parts supplied by these nerves, such postures not depending upon the condition of the nerve-centres.

Postures may be due to rigid muscular contraction dependent upon permanent brain lesion, such as descending sclerosis. Such cases should be kept separate from the postures due to a temporary, it may be momentary, condition of the central nerve-mechanism. It is hoped that some proofs have been given that the study of spontaneous postures as indications of the condition of the brain is useful, and that it may be considered as one of the exact methods of studying the nervous system in its physiological and pathological conditions.

The subjoined tables contain descriptions of eight typical postures applying the "Principles" to the study of each.

Postscript.

It seems desirable to give some further indication of the knowledge that it is hoped to gain from the systematic study of postures, and to give further descriptive details of the typical postures included in the tables. I do not attempt to indicate what special portions of the brain are concerned in producing different postures, and do not think such details can at present be given. I cannot localise a motor centre for the "hand in fright;" could we do so, it would afford some evidence as to what portion of brain mechanism is concerned in the mental condition called fright. In the case of an idiot, whose hands were usually in the convulsive posture, I took cast of the hands, and subsequently had the opportunity of examining the brain, which showed marked defects in the posterior convolutions. It is probable that difference in hand posture on the two sides indicates a different action of the two hemispheres, and of this I hope to give further evidence on another occasion founded upon the association of postures. When the hands are held free, a difference in the posture on the two sides, or different movements in the two hands, give

TABLE I.

Principles.	Nervous Hand.	Energetic Hand.	Hand in Rest.	Ortho-extended Hand.
1. "Anatomical analysis."	Wrist . . flexed. Metacarpo-phalangeals hyperextended. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . hyperextended. Thumb, internode hyperextended. Phalanges, relative position abduction.	Wrist . . . extended. Metacarpo-phalangeals flexed. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . . . flexed. Thumb, internode flexed. Phalanges, relative position adducted.	Wrist . . . flexed. Metacarpo-phalangeals flexed. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . . . flexed. Thumb, internode flexed. Phalanges, relative position adduction.	Wrist . . ortho-extended. Metacarpo-phalangeals ortho-extended. 1st and 2nd internodes ortho-extended. Thumb, metacarpo-phalangeals . ortho-extended. Thumb, internode ortho-extended. Phalanges, relative position adducted.
2. "Small parts contrasted with large parts."	Small parts characterised by hyperextension of the metacarpo - phalangeal joints in contrast with flexion of the large joint —the wrist.	Small parts in flexion in contrast with the flexion of the large joint—the wrist.	Small parts in flexion. Large parts in flexion.	Small parts ortho-extended. Large parts ortho-extended.
3. "Interdifferentiation."	Interdifferentiation marked, the small metacarpals and the thumb being extended in contrast with flexion of the large joint —the wrist.	Interdifferentiation marked, the small joints being all flexed in fingers and thumb in contrast with extension of the large joint—the wrist.	No interdifferentiation in the type described.	No interdifferentiation in the type described.

TABLE I. (*continued*).

Principles.	Nervous Hand.	Energetic Hand.	Hand in Rest.	Ortho-extended Hand.
4. "Collateral differentiation."	No collateral differentiation in the type described.	No collateral differentiation.	No collateral differentiation.	No collateral differentiation.
5. "Symmetry."	The type symmetrical; the two hands may differ.	Symmetrical.	Symmetrical.	Symmetry of this type complete.
6. "Excitation of weak centres."	Metacarpo-phalangeals extended, indicating excitement of weak centres.	Wrist extended, indicating excitement of the weaker centre of a large joint.	No extension of any joint, indicating no excitement of weak extensor centres.	Perfect balance, no excitation or weakness. This is the normal strong hand.
7. "General excitement or weakness."	Cannot apply here, as there is interdifferentiation.	Cannot apply here, as there is interdifferentiation.	General flexion indicating predominance of the strong flexor muscles from general weakness of the nerve-centres.	Perfect balance, no excitation, no weakness.
8. "Analogy."	Wrist flexed as in "hand in rest." Knuckles and thumb in hyperextension as seen in chorea, in subsultus tendinum in some irregular twitches in sleep.	Wrist extended as in "hand in fright." Knuckles and fingers and thumb flexed as in "hand in rest."	The type of complete relaxation of all the nerve-centres.	The type of complete balance of all the nerve-centres, hence the type of strength without excitement.
9. "Antithesis."	The energetic hand.	The "nervous hand."	α. The "ortho-extended hand." β. The "hand in fright."	α. The "feeble hand." β. The "hand in rest."

TABLE II.

Principles.	Convulsive Hand.	Hand in Fright.	Feeble Hand.	Ortho-extended Hand with Thumb Drooped.
1. "Anatomical analysis."	Wrist flexed or extended, &c. Metacarpo-phalangeals flexed. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . . flexed strongly. Thumb, internode flexed strongly. Phalanges adducted.	Wrist . . hyperextended. Metacarpo-phalangeals ortho-extended. 1st and 2nd internodes ortho-extended. Thumb, metacarpo-phalangeal . ortho-extended. Thumb, internode ortho-extended. Phalanges abducted.	Wrist . . . . flexed. Metacarpo-phalangeals flexed. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . . . . flexed. Thumb, internode . flexed. Phalanges adducted, metacarpus contracted.	Wrist . . ortho-extended. Metacarpo-phalangeals ortho-extended. 1st and 2nd internodes ortho-extended. Thumb, metacarpal bone adducted or drooped. Thumb, internode ortho-extended. Metacarpus straight, phalanges adducted.
2. "Small parts contrasted with large parts."	Condition of the wrist varies (it may be flexed, extended, adducted).	Small parts ortho-extended, while the large wrist joint is hyperextended.	Small parts in flexion. Large parts in flexion.	.. ..
3. "Interdifferentiation."	Interdifferentiation various; the wrist posture varies.	No interdifferentiation, all joints extended.	No interdifferentiation; all joints flexed.	.. ..
4. "Collateral differentiation."	Probably no collateral differentiation in most cases.	No collateral differentiation.	No collateral differentiation in the type described.	Collateral differentiation of the metacarpal bones, the thumb being drooped.
5. "Symmetry."	.. ..	.. ..	Symmetrical.	Not symmetrical; the metacarpus being straight but the thumb drooped.

TABLE II. (*continued*).

Principles.	Convulsive Hand.	Hand in Fright.	Feeble Hand.	Ortho-extended Hand with Thumb Drooped.
6. "Excitation of weak centres."	All the hand in strong flexion, but the wrist may be extended, indicating excitement of this weak centre.	All the extensor weak centres are excited.	No excitement of any weak centres.	No excitation of weak extensor centres, but marked weakness of the extensors of the thumb.
7. "General excitement or weakness."	General excitement of all the nerve-centres causes the flexors strongly to predominate.	General excitement of all the extensor centres indicating general excitement of the weak centres.	General weakness.	Perfect balance of nerve-mechanism for fingers and wrist, weakness of the centres for the thumb.
8. "Analogies."	.. ..	α. Analogous to the "energetic hand," with excitement of the weak extensor centres of the digits. β. Analogous to the "ortho-extended hand," with excitement of the weak extensor centre of the wrist.	.. ..	.. ..
9. "Antithesis."	Metacarpus contracted as in the "feeble hand."	Hand in rest.	Metacarpus contracted as in the "convulsive hand." Ortho-extended hand.	.. ..

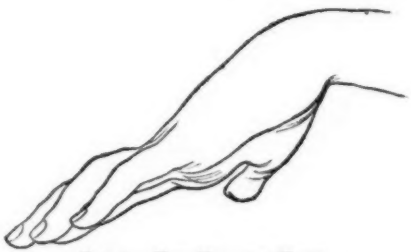


FIG. 1.—THE NERVOUS HAND.

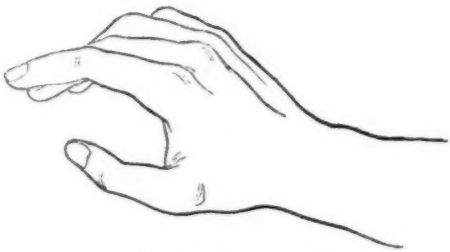


FIG. 2.—THE ENERGETIC HAND.

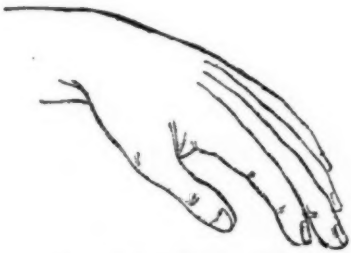


FIG. 3.—THE HAND IN REST.

FIG. 4.—THE STRAIGHT HAND.

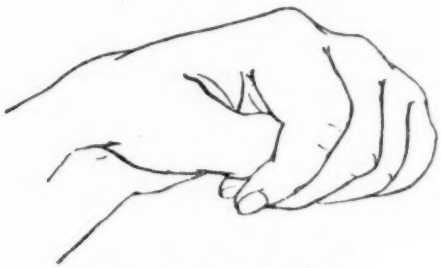


FIG. 5.—THE CONVULSIVE HAND.

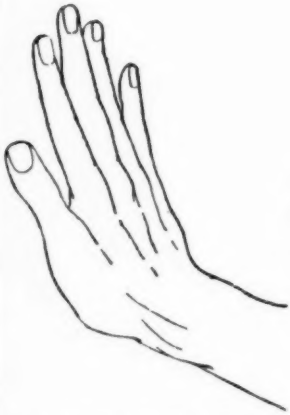


FIG. 6.—THE HAND IN FRIGHT.

FIG. 7.—THE FEEBLE HAND.

FIG. 8.—THE STRAIGHT HAND WITH THUMB DROOPED.

evidence that the two hemispheres are in a different condition. These studies show that in many people the nerve-centres are not strictly symmetrical in action, the average postures of the face and hands being asymmetrical, e.g., frequent one-sided, grinning, the nervous hand is often seen on one side only, usually the left; these observations may be conducted in healthy people, and give evidence of the individual's idio-synchronism. The study of postures gives examples of the application of the "Principles," enunciated above, and I think, affords some proof of their practical importance. Most of these points are commonplace clinical considerations, but principles vi. and vii. perhaps require further explanation and defence.

It may be readily admitted that it is desirable and important to know what are the motor indications of excitement or over-action of a nerve-centre, also to know the relative strength of the different motor centres, and what are the outward physical signs of wide-spread stimulation or weakness among portions of the nerve-mechanism. In numerous cases, where common experience has shown general strength, weakness, or wide-spread stimulation, etc., these principles of analysis have been applied, and some general knowledge has thus been obtained, but more exact knowledge is wanted. I trust by an experimental method, described in the 'Journal of Physiology,'[1] to be able to give hereafter a much more precise and definite account of these principles.

These studies afford evidence that some parts of the brain may be in a condition of weakness, while others are in a state of strength or excitement, and that one hemisphere may be weaker in action than the other. Again a change of posture may indicate a change in the corresponding nerve-mechanism, thus in chorea when the forearm is passively supported, we often see that the free hand passes from the posture of rest to the convulsive posture, thus indicating the change in the condition of the nerve-mechanism, corresponding to the muscles of the hand. By daily recording the most common involuntary postures of the free hand, we obtain indications of the daily condition of the nerve-mechanism corresponding.

[1] Vol. iv. No. 2, August 1883.

A few words as to the hand postures analysed in the tables.

The ortho-extended hand.—A strong man when asked to put out his hand, holds it out straight, all parts in the same plane, no flexion or extension, but perfect balance. This statement is founded on numerous observations. I have often requested a body of students, or other healthy subjects, to hold out their hands, so also healthy children, who could know nothing of my object, and thus I have had the opportunity of judging that this well-balanced posture indicates the normal action of the nerve-mechanism employed in the act.

The hand in rest may be seen in a strong and healthy subject during sleep, also when the hand hangs free and quiet, as when doing nothing, and in a state of mental quiescence. I have often seen it during quiet conversation, and in men while walking or travelling. The metacarpus is often slightly contracted, but not to such a marked degree as in the "feeble hand."

The convulsive hand.—This posture is well known. Trousseau described it in his 'Clinical Lectures' as commonly caused by the involuntary contractions in tetany. The condition of the metacarpus is specially noteworthy. I have taken casts of several cases and have measured many more. It is as Trousseau described it, arched or contracted by approximation of the metacarpal bones, which are screwed together as in the hand of the accoucheur. Important variations of this posture might be described; it is often seen in various pathological states.

The feeble hand.—If the disengaged hand of a feeble child or woman is held out we commonly see a slight, but important, departure from the hand in rest. The thumb is drooped, and its metacarpal bone is approximated to the palm, all the metacarpals being bent round as in the convulsive hand, this is a posture often seen.

Independent of all direct benefit that may follow from observing movements and postures, which are the result of the last movements, I maintain that all physical phenomena which are the direct outcome of brain-action are worthy of observation, record and analysis.

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MAY TABES DORSALIS SOMETIMES HAVE A PERIPHERAL ORIGIN?

BY HERBERT W. PAGE, M.A. CANTAB., F.R.C.S. ENG.

THE origin of the system diseases of the spinal cord is still profoundly obscure, notwithstanding that their clinical history and pathological changes have been carefully studied and recorded. Amongst them tabes dorsalis is one of the commonest, but its cause and origin are nevertheless wholly unknown. A history of syphilis is indeed often to be obtained from the victims of this disease, yet syphilis is far from being generally accepted as an undoubted cause, and were Duchenne de Boulogne now alive, it is improbable that he could materially alter the words which he wrote in his classical description of the malady many years ago: "Malgré le soin extrême que j'ai mis à étudier les antécédents des malades, je n'ai pu, dans quelques cas, entrevoir la moindre cause prédisposante ou déterminante." It is therefore not inappropriate perhaps to put the question which heads this paper, and even though the answer to it may be deemed inconclusive here, it may perhaps suggest a line of inquiry to other and more competent observers whose opportunities are greater than my own. I am still further emboldened to take the MS. out of the obscurity of a drawer, by the recent important contribution of MM. Pitres and Vaillard on non-traumatic peripheral neuritis, an account of which will appear in the "Abstracts of Foreign Journals."

Before attempting to answer the question, however, let me relate, and take as a text, the following case, that of a man who many years ago had "perforating ulcers" of the feet, and who now is the subject of tabes dorsalis, with a characteristic arthropathy of one joint.

F. G., aged 36, a spare, not unhealthy-looking man, presented

himself as an out-patient at St. Mary's Hospital on Jan. 5, 1882, complaining that for the last two months the top of his left foot had been much inflamed and somewhat painful in walking. The dorsum of the foot was found red and œdematous, and he said that he had been occasionally troubled with the same condition ever since his great toe had been removed seven years ago. He further stated that his toe had been lost from frost-bite in India, where he was then serving, and that in consequence thereof amputation had been thought necessary on his return home. He had had a chancre eighteen years ago, followed by rash, and in India he had had ague, but in other respects his health had been always good. The history being somewhat unusual, and the sole of his left foot being evidently somewhat anæsthetic, I asked him to show me his other foot, and with astonishment found that his right great toe was also absent. The following history was then obtained; that when in India, eight years ago, his right foot began to be bad with a corn under the metatarso-phalangeal joint of his great toe; that having cut the corn, it began to discharge, became so sore as to incapacitate him for duty, and on his return to England the toe was removed in the Netley Hospital. About a year elapsed, and his left great toe was attacked in the same way, speedily got worse and was amputated. Then it was that he was told his right great toe had been lost by frost-bite, an explanation of his malady which to himself was wholly new, although he adopted it afterwards. In reality, the condition had in both instances begun with corns which ulcerated deeply, so that, as he said, "a probe could be passed right through" the metatarso-phalangeal joints.

In his gait there was nothing remarkable, nothing in fact which might not fully be accounted for by the loss of his toes; but on testing him with his eyes shut, he was decidedly unsteady. The knee-jerk was completely absent on both sides, the vasti, however, contracting normally on percussion. The soles of his feet were unquestionably anæsthetic. The pupils, which were small and not quite equal, reacted very sluggishly to light. For the last two or three years he has had pains in the legs. He called them "jumping pains," and said they had been so bad as to make him call out. Under the influence

of hot fomentation to the foot, and the administration of iodide of potassium, the redness and œdema soon subsided, and he ceased to attend the hospital. I found him again on the 27th of February, 1883, when he told me that shortly after his last visit, or towards the end of January 1882, a corn formed under the fourth toe of his left foot, and that there followed a train of events which led to the removal of this toe in the Middlesex Hospital, he having failed to gain admission into St. Mary's. He was in the hospital for seven weeks, and was discharged well.

Some time in April 1882, a fortnight or so after he left the hospital, his left leg and foot began suddenly to swell as high as the knee. The swelling lasted for a week, and when it then subsided the ankle was left enlarged. This ankle has been swollen ever since, and there is now well-marked thickening, which is obviously due to bony enlargement of the lower end of the tibia and of the astragalus and os calcis. The ankle movements are tolerably free, and there is no bony crepitus. The ankle and foot are strangely widened, presenting very much the aspect of the foot of the patient who was shown by me at the International Medical Congress 1881,[1] and whose case has been more recently brought before the Clinical Society.[2]

The history of this joint attack so closely resembles that of the other case, in which both feet were affected, and unquestionably as symptoms or consequences of tabes dorsalis, that there can be little doubt of the ankle deformity being in this case also a true Charcot's arthropathy. The other phenomena remain the same.

There are few subjects at the present time of greater interest than the connection of such trophic changes as this case presented with central lesions of the spinal cord. Is the particular form of ulcer called "perforating ulcer" of the foot to be placed in the same category as the arthropathies which are not uncommon in the course of tabes dorsalis, or is it solely due to a peripheral neuritis which may sometimes precede, or even be a cause of, the special degenerative lesion of the spinal cord? Was this patient when he lost his big

[1] Vide 'Transactions,' vol. i. p. 124.
[2] 'British Medical Journal,' vol. i., 1883, p. 772.

toes the subject of tabes dorsalis, or is the tabes which he now has but a later consequence of the peripheral disease to which his perforating ulcers were originally due? In the exhaustive paper on perforating ulcers which they brought before the Royal Medical and Chirurgical Society in 1879,[1] Mr. Savory and Mr. Butlin remarked that, "if called upon to express an opinion of the nature of the disease, we should say that the so-called ulcer is the result of pressure, or violence, or injury to structures, whose nutrition is impaired, or whose vitality is defective from disease or degeneration of the supplying nerves. In proof of this we would draw attention," they write, "to the fact that marked changes have been discovered in the nerves passing to the affected part in every case in which they have been thoroughly examined." Professor Ball and Dr. Thibierge, however, see in some cases, though not by any means in all, a more central cause than degeneration of the peripheral nerves, and in the paper read by them before the International Medical Congress in 1881, they come to the conclusion[2] that "perforating ulcer of the foot is unquestionably connected, in a tolerably large number of cases, with the earlier or later periods of tabes dorsalis," presumably, that is, with the *cord* degeneration which is peculiar to this disease. That there may be, however, other forms of "perforating ulcer" is acknowledged by these authors, and examples thereof have been recorded—to refer only to recent contributions—by Mr. Treves in a paper on "Perforating Ulcer of the Foot, and progressive Locomotor Ataxia," in the 'Lancet' of Oct. 21, 1882. He relates a case in which the original ulcer of one foot "made its appearance before any evidences of ataxia had developed;" and doubting, therefore, the causal relationship in that case between the ulcer and the disease of the spinal cord, he refers to three other cases under his observation during the last few years, where there were neither anæsthesia, nor undue sweatings of the foot, nor any evidence of nerve affection of any kind. He urges that "perforating ulcer" is a purely local affection, produced by purely local causes, which would act with increased vigour upon a part whose vitality is in any way impaired, but that

[1] 'Trans.' vol. lxii. p. 382. [2] 'Trans.' vol. ii. p. 55.

that impairment is not of necessity dependent on disease or degeneration of the supplying nerves. In other words, he would not regard the ulcer as a trophic change.

There is, doubtless, no inherent reason why an ulcer of this particular kind should not be dependent on local causes only, set up by the long-continued and painful pressure of a corn on the soft parts beneath it, by consequent inflammation and suppuration, and by the deep burrowing of pus in the line of least resistance, so as ultimately to reach the bone. The association, however, of this form of ulcer with some degeneration of nervous structures is too frequent not to raise a suspicion that there is indeed some closer bond between the two. In the case with which this paper was opened there was in all probability nothing at the time when the patient lost his toes to suggest the existence of central disease, and five years went by before he began to have those jumping pains which M. Charcot and others have clearly recognised as being amongst the very earliest symptoms of true tabes. M. Charcot, it may be remembered, has even suggested that the first period of tabes dorsalis should be called the "period of lightning pains," because they "may of themselves alone compose the entire symptomatology of the disease." In Mr. Treves' case, also, a period of five years elapsed before his patient showed undoubted symptoms of tabetic degeneration of the spinal cord; and several other cases have been recorded where perforating ulcer was coincident with lightning pains, or even preceded their onset by a considerable interval of time.[1] Such observations as these invite the question whether perforating ulcer may not sometimes be an early and premonitory sign of a disease whose more telling symptoms are not yet developed, and whether the degenerative changes in the spinal cord may not even be an extension, or consequence, of those changes in the peripheral nerves which MM. Savory and Butlin assure us have been discovered in every case in which they have been thoroughly examined.

Grave pathological changes in the nerve-centres may, it is

[1] See a case recorded by Duplay and Morat, 'Arch. Gén. de Méd.' 1873, p. 268; one by M. V. Hanot, 'Arch. de Physiologie,' 1881, No. 1, p. 157; and one by Mr. F. A. Southam, 'British Medical Journal,' vol. i., 1883, p. 1222.

well known, be originated by extension of disease from peripheral lesions of nerves, or by long-continued irritation of nerve-trunks, as after amputation of a limb.[1] Several cases of the kind have been recorded; and the fact that perforating ulcer appears not unfrequently to be an early lesion in cases which ultimately show the true symptoms of tabes dorsalis suggests this important question, whether the degenerative changes in the spinal cord are not in such cases a direct consequence of the changes in the peripheral nerves? True, a weighty objection to this theory seems to present itself in the fact that the lesion of tabes dorsalis is almost strictly limited to the posterior parts of the spinal cord; but the objection loses weight when we call to mind that, as Savory and Butlin point out,[2] "the symptoms of nerve lesion in all cases point to affection of sensation and of nutrition, rarely to affection of motion; and never to affection of motion without affection of sensation or nutrition. In other words, the sensory and nutrient, or vaso-motor nerves, are at fault, the motor nerves generally unaffected." And, speaking of the microscopical changes, they go on to say: "It is of course impossible in the transverse section of a mixed nerve to decide whether any individual fibril is motor or sensory or nutrient (*i.e.* vaso-motor, or perhaps trophic). But, inasmuch as the fibrils which compose the sympathetic nerve are for the most part small, and either non-medullated or almost without medullary sheath, it may be supposed that nutrient fibrils in mixed nerves bear similar characters, and that a certain number, therefore, of the smallest fibrils seen in a transverse section are nutrient. . . . And although it does not appear to be absolutely true," that sensory fibrils may be distinguished from motor fibrils by their thinner medullary sheaths, "inasmuch as motor nerves contain thinly-medullated fibrils, and sensory nerves contain some thickly-medullated fibrils, there is generally so distinct a difference that we can recognise with ease a motor or sensory nerve. The fibrils of the former are larger, more thickly

[1] See Weir Mitchell's "Injuries of Nerves," and a recent case recorded by M. Charcot, 'Progr. Méd.' 1883, p. 157, *et seq.*; and Dr. Ferrier's paper on "Hemiplegic Muscular Atrophy of Peripheral Origin," 'Brain,' Vol. V. p. 521.

[2] *Op. cit.* p. 383.

medullated, and more uniform in appearance. We believe, therefore, that the smallest fibrils of a mixed nerve are chiefly nutrient and sensory, and that the larger fibrils are chiefly motor. The histological characters of the nerves in the two cases examined by us strengthen this belief. Both are characterised by absence of, or damage to, or obliteration of, the small fibrils, either from defect, which is perhaps congenital, or from disease, whilst the larger fibrils remain apparently intact." The defects, therefore, of sensation and nutrition, which are often observed in cases of perforating ulcer, and of which latter the ulcer itself is one of the most striking symptoms, seem to these authors to be "due to absence or degeneration of the sensory and nutrient fibrils of the supplying nerves." And if it be true that these fibrils are especially picked out for damage, whether primarily from some unknown cause, or as the result of mechanical pressure or constant irritation of the more delicate constituents of a compound nerve, it does no violence either to physiology or pathology to assume that in its centripetal march the degeneration should involve those parts of the spinal cord which are in most immediate connection with the sensory roots. The essential degeneration of tabes dorsalis is limited to the sensory tracts of the spinal cord; as Leyden puts it, tabes is "eine Erkrankung der sensiblen, d. h. der centripetal leitenden Faserelemente," and there is neither more nor less difficulty in accounting for a degeneration being limited to one portion only of the spinal cord than to one set of fibres only of a nerve. And whether seated in the one part or the other, whether in spinal cord or in compound nerve, the degeneration will follow a true physiological—in this instance centripetal—march. Pathological investigation has yet to show us where that march begins, and I would urge a much more extensive inquiry into the morbid appearances of the peripheral nerves in all cases of tabes dorsalis which come to the post-mortem table. "In der Regel nimmt die Intensität der Rückenmarks-Erkrankung von unten nach oben ab und ist im Lendenmarke am intensivsten; doch giebt es auch Fälle, wo das Lendenmark relativ wenig, dagegen der mittlere Brusttheil, oder gar die Cervicalanschwellung viel

stärker ergriffen sind."[1] What is the explanation of the rule that the lumbar portion of the spinal cord is most frequently and most severely affected with the degeneration of tabes? Is it impossible that the explanation may sometimes be found in degeneration of those peripheral paths of the nervous system which may be far more exposed than are the central paths to unnatural and morbid influences?

At any rate we have these facts before us; that perforating ulcer is a symptom or consequence of some nutritive derangement of the affected part, and that in a goodly number of cases tabes dorsalis has been detected some time after this symptom of peripheral nerve-lesion existed. And it seems not altogether out of the question that in such cases the disease may have really begun at the peripheral parts of the general nervous system; that the nerve lesion was originally caused by the continued painful—often severely painful—pressure of the corns, by the protracted sensory disturbance giving rise to ultimate structural change, and that in the course of time—now shorter, now longer—the degeneration, travelling upwards, reached the spinal cord, and then, and only then, gave rise to the symptoms of tabes dorsalis. The question can only be solved by the careful observation of cases of perforating ulcers over a long period, that it may be known whether in any considerable number of them tabes dorsalis is ultimately developed, there having been originally no symptom of this central disease.

If such a sequence of events be clearly established, it will go a long way to support the view which, not unmindful of many objections, I have here ventured to put forward, that tabes dorsalis may in some cases have a peripheral beginning, and that a painful corn under the metatarso-phalangeal joint is a by no means trifling affection, little deserving of treatment. Present facts are suggestive; future observation can alone give conclusive answer to the question "May tabes dorsalis sometimes have a peripheral origin?"

[1] Leyden, 'Real-Encyclop.', Bd. xiii. p. 366, 1883.

NOTE ON THE PATELLAR KNEE-JERK.[1]

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In view of the uncertainty at present existing with regard to the physiology of the so-called patellar reflex, I venture to record a few observations on the condition of this phenomenon when the subject of experiment is under the influence of nitrous oxide gas.

In 1881, while experimenting (on myself) with this gas for a different purpose, it occurred to me to contrast the conditions of the superficial and deep "reflexes," taking the plantar reflex as an example of the former, and the patellar phenomenon to illustrate the latter. Being aware of the fact that deep chloroform-narcosis abolishes all the "reflexes," superficial and deep, and yet that with ether-narcosis, however deep, ankle-clonus frequently appears, I was scarcely surprised to find that the knee-jerk persisted in the deepest anæsthesia from nitrous oxide, while the superficial reflexes all disappeared. This distinctive difference between the two groups of "reflexes" was so striking, as to merit further observation, of which the following is a brief summary.

The necessity of estimating exactly the degree of consciousness in dealing with an anæsthetic reagent, such as nitrous oxide, is obvious when the statements of patients (who have been operated upon while under its influence) are taken into account. To avoid the possibility of error in stating the depth of narcosis, only the result of experiments (fifteen in number)

[1] I desire here to express my obligations to my friends, especially to Mr. Henry Maudsley and Mr. Halliburton, for kind help in making the above observations.

made on myself is here stated, but the facts were verified by observations made on other subjects.

In all cases the anæsthesia produced was complete; after the usual symptoms attending the commencement of the administration of the pure gas were fully marked, absolute unconsciousness followed in from 90 to 120 seconds.

The anæsthesia was pushed until rigidity and sometimes cyanosis resulted. The recovery of consciousness was very frequently attended with considerable muscular spasm and semi-co-ordinated convulsive struggles and excitement. The "reflexes" were examined at regular intervals of five seconds from the commencement of the experiment to the return of consciousness; the sole of the foot being pricked with a pin, for the plantar reflex, while the opposite patellar tendon was struck by a stethoscope or the hand. (The knee-jerk being normally very well marked, there was no opportunity for error of observation in eliciting the phenomenon, and the plantar twitch was also very active.)

Result.—In the deepest narcosis the condition of the reflexes was as follows:

Superficial.—Plantar and conjunctival abolished.

Deep.—Knee-jerk persistent.

Thus it is seen that although the whole central nervous system, viz. the encephalon and spinal-cord, was poisoned by the gas, the knee-jerk still continued active.

Taking this fact in connection with the short time expended in the knee-jerk contraction, the conclusion is unavoidable that the shortening of the rectus is due to local irritation of the muscle from sudden elongation, the integrity of the normal tonus being maintained. It seems scarcely necessary to refer to this latter point, but in an admirable paper on this subject,[1] Dr. Prevost has shown, that in rabbits, anæmia of the lumbar enlargement of the spinal-cord (produced by compression of the abdominal aorta) is followed by the simultaneous (?) disappearance of the deep and superficial "reflexes." It would appear, however, that in nitrous oxide we have a reagent with which it is possible to perform the crucial experiment of

[1] Published in the 'Revue Médicale de la Suisse Romande,' Jan., Feb., March 1881.

suppressing the superficial reflexes without abolishing muscle tonus. With regard to the different results obtained by using different anæsthetic agents, the following conclusions may be drawn from the researches of Eulenburg,[1] also confirmed by Prevost's experiments with ether, &c., viz. (1) that the deep "reflexes" are only abolished by the denser anæsthetic reagents, such as chloroform, &c., and are exaggerated by the more volatile reagents, such as ether, &c., but that both superficial and deep "reflex" action is completely abrogated in the asphyxial state; and (2) that in the latter condition, whether induced mechanically, or by drugs such as nitrous oxide gas, the superficial disappear before the deep "reflexes."

[1] 'Centralblatt für Med. Wissensch.' No. 6, 1881.

Clinical Cases.

A CASE OF EPILEPSY—AURA OF TRUE VERTIGO WITH AUDITORY SENSATIONS—VAGUE COMPLEX REMINISCENCES—EMOTION OF HELPLESSNESS—SPASM PRIMARILY LEFT-SIDED.

BY CHARLES MERCIER, M.B. LOND., F.R.C.S.

LIZA ANN A., æt. 49, married; three children living; three dead. No miscarriages.

No family history of neurosis, save that a niece of patient's had puerperal insanity. Patient persistently attributes her illness to "change of life." Her health was good until Christmas, 1881. At that time, while walking in the street, she had the first of a series of attacks, which have recurred at intervals ever since. In this attack she felt giddy, reeled about, and fell. She felt "as though she was going to lose her senses." She had to catch hold of something, and then fell down. She felt as if she was going round. This feeling of going round was, she states in answer to questions, "like a dreadful giddiness." When asked in which direction she went round, she alters her position, and appears to make a careful effort at remembrance. She then says positively that she seemed to go round from left to right. She is very positive that this was the direction in which she appeared to turn, and she adheres to this statement when repeatedly cross-questioned at different times. During the onset of the attack she had other feelings. She is not clear as to whether these feelings existed at the same time as the vertigo, but it would appear as if sometimes these feelings and sometimes the vertigo was the preponderating sensation at the outset of a fit. The feelings she describes as follows: it is "as if I was going a long distance;" "as if I was going further off;" "as if I was going right out I don't know where;" "once I felt as if I was going down into a pit;" on another occasion the feeling was "as if there was something in front of me and it broke off, so that I could not get through it; I tried and tried, and could not get through it." On another occasion this feeling "came on as I was taking the kettle off the fire, and I had to put the kettle

down and run out of the room and catch hold of the banisters." She states that the attacks are accompanied by strange feelings in her ears. She volunteers the statement without being questioned, "that they are not in her head so much as in her ears. It is, she says, "as if something was going right through her ears." On one occasion she heard a loud bang in her ears, "as if a tea-tray had been struck a violent blow." (She has no defect of hearing with either ear.)

After she fell down in an attack she "lost her senses altogether;" but she subsequently modifies this by saying that she could hear that people were talking around her, and could distinguish what they said. During many attacks she was conscious throughout, and knew that she was "like this" (here she imitates most accurately the facial movements of a convulsion beginning in the left side of the mouth and affecting the eyes). She states, without questioning or prompting of any kind, that the left eye worked much more than the right, and she imitates the characteristic twitching with remarkable fidelity. She goes on to say that her arms also would work about; and here she imitates fairly well the movements of bilateral clonic convulsion. The legs, she says, were not affected so badly as the arms; but sometimes they would shake about, especially the left leg (*n. q.*), and here she imitates the movement of clonic convulsion in the left leg. She occasionally—but not, she thinks, during a fit—has a feeling going down the arm; she particularises the left arm, but says, in answer to questions, that she feels it in both. She is certain that it goes *down* the arm, sometimes beginning in the shoulder, and sometimes in the elbow. She says it is like a powder going down the arm, and at the end a feeling comes at the tips of the fingers like pins and needles in them. After a fit she feels "very bad all over," and "as if her feet were in dough."

To direct leading questions, she says that she never felt angry during nor after a fit. She is right-handed.

Remarks.—The foregoing record is a contribution to our knowledge of a class of very obscure and recondite cases, to which attention was first directed by Dr. Hughlings-Jackson in Part X. of 'BRAIN,' and resembles in many respects a case that I shortly afterwards contributed to these pages in illustration of his views. From the point of view posited in Dr. Jackson's article, the present case is very interesting and important.

The main conclusions arrived at in the article referred to—conclusions which were, however, avowedly tentative and provisional—were: (1.) That all "warnings" of fits, in other words, all mental states occurring at the outset of a fit, may be divided into two classes, according as they are developments, however crude, of objective or of subjective sensations; by

objective sensations being understood those feelings which normally occur during reaction of the organism on the environment; and by subjective sensations, those which occur during action of the environment on the organism. (2.) The left cerebral hemisphere is that which represents the most objective movements—those movements which are concerned in primarily operating on the environment; and is that during action of which the objective sensations arise. To this order belong the movements concerned in orientation, whose disorder is accompanied by vertigo; movements of the right hand, which is the leading member in acts on the environment; the movements of speech, which is "a process by which are symbolised relations of things in, or as if in, the environment; that is, things considered objectively, and whose disorder is aphasio." (3.) The righ themisphere, on the other hand, is active during sensations of the subjective order, and its activity gives rise to the answering movements. It is active during sensations of smell and taste, during hunger, one of the most subjective of all sensations; and its activity gives rise to the answering movements of mastication, deglutition, spitting, &c. Feelings of colour, the subjective side of the function of vision and of hearing, are also connected with the action of the right hemisphere.

If these suppositions are correct, we must expect to find that when vertigo occurs at the outset of a fit, or when aphasia occurs among the sequelæ, or when an emotion belonging to the objective series is aroused, the accompanying spasm will be primarily right-sided, all these occurrences being manifestations of the action of the same hemisphere—the left. On the other hand, "warnings" of coloured vision, of smell, of noises in the ears of the epigastric sensation, together with emotions of the subjective order, should be accompanied by left-sided spasm, since this occurs from, as those occur during, action of the right hemisphere.

The "complex vague reminiscences," or "dreamy states," such as occurred in the foregoing case, are, in Dr. Hughlings-Jackson's experience, mostly associated with warnings of the "subjective" order, and with left-sided spasm; and the warnings in such cases are not only of the subjective order, but are of the most strongly pronounced subjective character;—such warnings as those of smell, of the epigastric sensation, and presumably of taste. It is rare in his experience for the dreamy state to attend warnings of coloured vision and noises in the ear, and he had at the time of writing (1880) taken notes of only one case in which this state occurred with the warning of definite or true vertigo. Although Dr. Jackson states that this dreamy state is usually associated with spasm

beginning on the left side, it would appear that this opinion is merely the epitome of his experience, experience which, considering the rarity of the cases, may easily prove exceptional; and in any case the validity of his inferences is not in the least impaired by the occurrence of these states in association with the objective warning of vertigo, as in the present case, and as in the previous case that I published. I believe that it is not the occurrence, but the character of the reminiscence, which is associated with spasm of one or other side.

Turning now to the case here recorded, we find that in some respects it is in harmony with Dr. Jackson's reasonings, in some respects it is anomalous with regard to them, and in some respects it is exceptional to his experience. It is exceptional to his experience, as already pointed out, in the association of the "dreamy state" with true vertigo and noises in the ear. It is at variance with his conclusions in so much that vertigo, a feeling belonging to the objective order, is associated at the onset of the fit, not only with noise in the ears, a feeling of the subjective order, but with spasm primarily left-sided, that is, with action of the right hemisphere. Again, the vertigo was of such a character that the patient felt as if she herself was turning from left to right, which is equivalent to the displacement of objects from right to left; which is in harmony with the commencement of the spasm on the left side indeed, but is opposed to the hypothesis that the vertigo occurred during discharge of the left hemisphere.[1]

On the other hand, the points as to which this case harmonises with Dr. Jackson's expectations are much more numerous and are very important. The "dreamy state," or reminiscence, did, it is true, accompany vertigo, a feeling of the objective class; but then it is a striking fact, and one which strongly corroborates Dr. Jackson's notions, that *this reminiscence was itself of a markedly objective character*. Vertigo is a motor disorder—a disorder of a group of highly representative movements or motor arrangements; and, correspondingly, every one of the vague complex mental states which in this case occurred together with, or in connection with, the vertigo, was a reminiscence of *movement*. "As if I was going a

[1] After the above paragraph was in the press, a more careful perusal of Dr. Hughlings-Jackson's article convinced me that I had greatly exaggerated the discrepancies between his conclusions and the facts of this case. The vertigo which he believes to be accompanied by right-sided spasm, and to signify discharge of the left hemisphere, is that vertigo in which there are *apparent movements of objects to one side* (the italics are Dr. Jackson's). Vertigo such as occurred in the present case is explicitly distinguished by him from the form of vertigo which he believes to accompany right-sided spasm, and to be so rarely associated with the dreamy state. The distinction is so clearly and repeatedly stated that I am at a loss to know how I can have overlooked it. I can only offer the explanation given by Dr. Johnson in a similar case,—"pure carelessness."

long distance," "as if I was going further off," "as if I was going right out I don't know where;" these are the patient's expressions; and in every case the idea is one of movement, not initiated by, or connected with, anything happening in the environment, but of movement spontaneously initiated by the organism.

These reminiscences, of which the above is the cognitive aspect, are accompanied by feeling; and the feeling, which is strongly pronounced, is of a strictly corresponding order—the objective order. Dr. Jackson considers anger an emotion of the objective order, and contrasts it with fear, which he gives as an example of a subjective feeling; but it seems to me that anger as well as fear must be considered subjective in the sense in which he uses this term, of "answering to what is physically the effect of the environment on the organism." Anger, no more than fear, can arise without some circumstance in the environment first acting to elicit it. And not only are both anger and fear primarily initiated by the action of some agent in the environment upon the organism, but in both an equivalent reaction takes place. It is true that the reaction in one case takes the form of counteraction, while in the other it takes the form of avoidance; but it seems to me that a dog running away from a bigger dog displays as energetic a reaction as the same dog fighting a smaller one; and the accident of the size of the object cannot surely make a difference in the *order* of the feeling that it evokes. Feelings of the objective order, properly so called, appear to me to be not those which accompany movements that are reactions in any proximate sense on the environment, but those which accompany actions primarily, spontaneously, and *proprio motu* initiated by the organism. A simple example of such feelings is Effort, and among the more abstract and highly evolved feelings of the same order are the emotions of Power and Helplessness. The latter is the feeling which accompanied the other manifestations of irregular objective activity experienced in the case under consideration. The patient states that she had a feeling "as if there was something in front of me, and . . . I tried and tried, and couldn't get through it." Evidently here was a vague but massive feeling of helplessness, a feeling which is, as has been said, of strictly objective character.

Not only was the cognitive aspect of the reminiscences of a markedly objective character; not only was the accompanying emotion of an order strictly objective; but the vertigo, which is always an objective manifestation, had in this case the character of ultra-objectivity. For the giddiness was not an experience of things going round, not, that is, a sensation cognised as being initiated by things in the environment; but it was

described by the patient to be as if *she herself* were going round; that is to say, it accompanied the activity of motor processes that represent spontaneous movements. It is remarkable that in the only case that Dr. Hughlings-Jackson adduces as experiencing the dreamy state with definite vertigo, this dreamy state was one conspicuously objective in the sense here used. It was a feeling purely though crudely motor, —a feeling of being in some other place—several places, not any particular place—that is to say, it had not enough of the subjective or environmentally-initiated element to give it even the vaguest localisation.

Lastly, my patient has, with the other mental accompaniments of spasm, auditory sensations more or less definite, or at any rate feelings referred to the ears, and these feelings belong not to the objective but to the subjective class. Knowing how closely the ear is associated with the function of orientation, we must not be surprised at this, although in some respects it is in conflict with the suppositions made.

To summarise the facts of this case. There occur at the outset of the attack (1) true vertigo, a sensation always of the objective order, and in this case ultra-objective; (2) a vague representation of spontaneous movement,—a cognition of the objective order; (3) an emotion of helplessness,—also of superlatively objective nature; (4) an absence of all emotions of the subjective class; (5) auditory sensations, which, though primarily of the subjective order, are yet, regard being had to the vertigo, not altogether anomalous; and (6) these conditions, all connected together by one underlying similarity into a coherent group, are associated (in a right-handed person) with spasm primarily left-sided, an association altogether exceptional, although not perhaps altogether inexplicable. There is at least a definite preponderance of the spasm on *one* side, and although this side happens to be the wrong side, according to the hypothesis, yet in occurrences of such extreme complexity, we must be thankful for any definite association of them, even if it make matters at first more puzzling than before.

It should be stated that, as will be seen from the foregoing, the connotations attached herein to the terms "objective" and "subjective" are not absolutely identical with those used by Dr. Jackson, although they are closely similar.

A CASE OF HYSTERICAL (?) RAPID BREATHING IN A YOUTH.

NOTES BY M. W. H. RUSSELL,

Resident Medical Officer, Royal United Hospital, Bath.

CHAS. E., aged 19, single, paper-hanger, admitted into Duncan Ward on December 27, 1882, under the care of Dr. Fox.

There is no history of any nervous disorder in the family; no history of phthisis, and none of rheumatism or chorea.

Patient had measles, scarlet fever, and pneumonia at the age of 3 years; was not ill again until 13 years old, when he had "low fever." Never had fits. He recovered well from the low fever, and with the exception of some shortness of breath (panting after any unwonted exertion), which appears to have first manifested itself about this time, he remained fairly well until two years ago.

Two years ago the shortness of breath became more marked, and he attended as an out-patient at this hospital, and at a dispensary in the town, without deriving much benefit. At this time the shortness of breath was not attended by any unusual symptoms.

It is only during the past six months that the dyspnœa has acquired its present form. For that time he has been entirely precluded from work, on account of attacks of extremely laboured breathing, complicated with twitchings of face-muscles and spasmodic jerkings of limbs, and other nervous phenomena. Any excitement greatly aggravates the symptoms. At the end of attacks, patient for the last six months has been noticed to "bark like a dog."

Patient is a thin, pale, emaciated, dark-haired youth. He is in a most distressing condition. He has paroxysms, occurring at intervals of about 15 seconds, of forced breathing, in which he sets all his extraordinary respiratory muscles to work. He cannot lie down, and though much exhausted when the paroxysm comes on, he leans forward, fixing his hands on the bed, so as to give a *point d'appui* for his pectoral muscles,

which, together with all muscles which can aid respiration, are brought into requisition. The shoulders move up and down, the alæ nasi are hard at work, the mouth is held half open.

Each paroxysm consists of about 20 to 30 forced and hissing inspirations; each successive inspiration is deeper than its immediate predecessor, the last one being apparently the acme of his inspiratory power. The series is ended up by a peculiar rough, barking noise, several times repeated, as though he were attempting to hawk up some very viscid secretion in his large tubes. Nothing is however expectorated. The noise was mistaken by a physician in the ward for a carpenter sawing wood. The paroxysms are not followed by any period of apnœa, as he breathes quite tranquilly and evenly in the intervals, which are rarely longer than 15 seconds in duration. He has a peculiar furtive, helpless and alarmed expression of face, more marked during the actual paroxysm. When spoken to, he exercises considerable control over himself, and checks the seizures sufficiently to be able to answer clearly and intelligently any questions; but when not actually speaking, his distressing condition is immediately re-established.

Chest.—Long and narrow, walls emaciated, lower spaces very concave; expansion movements almost entirely eclipsed by elevation movements, which are much exaggerated. Area of pulmonary resonance is increased in all directions, and the note itself is fuller than usual. Full resonance over upper sternum. Cardiac dulness obliterated, and upper level of hepatic dulness depressed about 1½ inch.

All over chest the loud hissing laryngeal sounds are heard conducted, masking all other sounds during the attack; during the intervals the normal breath-sounds are obscured by sonoro-sibilant râles. Expiration is much prolonged.

Heart.—Apex felt feebly beating in normal situation. Apex sounds muffled. Basic sounds very distant. No bruit heard at base or apex.

Abdomen.—Liver felt 1 inch below costal margin, spleen not felt, no dulness made out.

Urine.—Acid, s. g. 1020; no albumen, no sugar. T. 99·6: P. 76.

Ordered a mixture containing: Liq. sod. arsen. ♏ iv; chloral hydr. gr. xx; potass. bromidi gr. xx aq. ℥i, *ter die.*

The pulse is not found to vary materially during the respiratory explosions. It is occasionally a little accelerated.

Dec. 28th.—Patient remained in much the same condition all through yesterday afternoon. Towards evening, after the second dose of his medicine, he began to get drowsy and early

fell asleep. He slept well and soundly, and breathed quite easily and quietly. Immediately on waking, the peculiar rhythm of respiration was re-established, but not with such painful intensity, the intervals being a little longer (20 seconds), and the breathing itself less forced. T. 98·2: P. 92. The sucking in of lower intercostal spaces is very marked during the laboured breathing. Patient exhibits slight choreic symptoms when watched; the brows are knit, the mouth pursed, the eyelids twitched. Bowels open. E. T. 99. Patient remains same. Sleepy.

Dec. 29*th.*—T. 98·8: P. 96, of fair quality.

Patient has had another good night and has since been dozing. Is perfectly quiet when asleep. Average length of intervals to-day 20 seconds; the paroxysms themselves consist of 15 forced inspirations. Patient has greatly recovered from his exhaustion. He can ward off seizures longer when talking. The barking is less harsh than it was.

E. T. 98·6: P. 72. Has dozed a good deal during the day. Is easily awakened, and immediately the respiratory phenomena recur. Each paroxysm now consists of ten rapid forced inspirations on an ascending scale, i.e. each one increasing in depth. The whole are performed in about 19 seconds.

The barking is to-day very occasionally omitted.

Dec. 30*th.*—T. 98. Patient is picking up well in his general health. The intervals are not much prolonged, still averaging about 25 to 30 seconds, but the respiratory efforts are less distressing and less panting.

Ordered to continue mixture, with the addition of Liquor atrophiæ sulph. ♏ 1½ to each dose.

Dec. 31*st.*—T. 98·6. Complains of throat feeling dry. Pupils are slightly dilated. E. T. 98·8.

Jan. 1*st.*—T. 99. Has had a very good night. Takes food well. Fits continue to diminish in severity, and bark is only once, twice, or at most thrice repeated after each. It is also less harsh. E. T. 98.

Jan. 2*nd.*—T. 98. Improvement maintained, but very slow. Seems fairly comfortable. Whenever awake, patient does not escape. The fits come on every 20 or 30 seconds, but are now reduced to four or five panting respirations and a bark. Sleeps and eats well. E. T. 98.

Jan. 3*rd.*—T. 98: P. 80. Small and feeble. The pulse is to-day found to slow slightly at the commencement of each attack. These remain of only moderate severity, but the intervals are most irregular, and are often as short as 5 or 6 seconds. Barking less. Throat very dry.

Mixture to be taken only twice a day.

Jan. 4*th.*—T. 98. Patient seems disturbed this morning.

Breathing more jerky; more barking. Paroxysms increased again to 8 or 10 breaths.

Jan. 5th.—T. 98·6. More excited. Intervals only 6 seconds. Paroxysms 15 or 20 breaths. Slept well.

Ordered Santonin, gr. iii; calomel gr. iii; to be taken at once, and to be followed by ol. ricini ℥ss. *cras mane.*

Jan. 6th.—T. 98. Slightly quieter. Bowels open. No worms expelled.

Ordered, Liq. arsen. ♏ij; pot. brom. gr. xv; chloral hyd. gr. xv, t. d. s.

Jan. 7th.—Much quieter again. Paroxysms less frequent. Has intervals of 4 or 5 minutes. Patient says that as the attacks become less frequent, his breathing feels more oppressed.

Jan. 8th.—Paroxysms rather longer (20 or 30 breaths). More barking; choreic twitching more marked.

Jan. 9th.—Quieter. Mixture omitted.

Jan. 10th.—Much more unruly again; scarcely ever free from forced breathing. Feels giddy after fits. Bowels open.

Ordered Zinci valerian. gr. vi, in pil. t. d. s.

Jan. 11th.—Distinctly worse. Intervals reduced to 6 seconds. Fits altered in character, consists of about 30 inspirations, culminating in a hiccough, after which one bark. At times the intervals are abolished, and the forced breathing is kept up for over a minute.

Replaced on Pot. brom., chloral and atropia.

E. T. 98·6. Ophthalmoscopic examination revealed nothing abnormal in the fundus of either eye. It was noticed that when the patient's attention was concentrated upon the ophthalmoscope, the respiratory spasms were held in check.

Jan. 14th.—Very much quieter.

Jan. 28th.—Been gradually improving since last note.

Feb. 10th.—Still slowly improving.

Feb. 28th.—Discharged. As soon as the medicine is stopped, the fits return.

March 20th.—Came as out-patient. Has been taking medicine regularly since discharge, and has enjoyed almost entire freedom from symptoms.

April 2nd.—Came again as out-patient. Still finds old symptoms return as soon as medicine is withheld. Is very anæmic. Has a few very mild attacks still daily.

August 10th.—The patient presented himself again to-day. His symptoms still persist, becoming much worse if the medicine is left off for more than two days. Whilst taking the medicine, they are kept more or less in abeyance.

CASE OF PRIMARY LATERAL SCLEROSIS OR SPASMODIC TABES.

BY JOHN HOPKINS, F.R.C.S., ENG.

Medical Superintendent, Cleveland Street Sick Asylum, W.

J. M., aged 21, porter, was admitted August 14th, 1881. Father and mother living, and stated to be in good health. All brothers and sisters, thirteen in number, died under four years of age, two of them of smallpox.

Patient had fits when a child. Never had venereal disease, smallpox, or any illness till twelve months ago, when he got wet, and within a week found his legs becoming weak and dragging, and his gait tottering; he improved a little, but the weakness and tottering never left him entirely.

The following was the condition on admission. Patient is fat but pasty-looking, and feels in perfect health. No signs of congenital syphilis. Gait peculiar, rolls from side to side, and as each limb is brought forward it jerks irregularly, and when put down stiffens and trembles. No difficulty in standing with eyes closed. On tip-toe the legs tremble. Percussion of the patellar tendon causes not only heightened patellar reflex, but trembling of the limb. Ankle clonus marked. Slight loss of sensation in the sole of the right foot (?) No wasting of muscles. Arms not affected. No symptoms referable to special senses, tongue, stomach or bladder. Fundus oculi normal. Heart and lungs free from signs of organic lesion. Urine clear and free from albumen.

Patient for some months got up daily, ate and slept well, and expressed himself as feeling perfectly well, but walking became increasingly difficult, and all the symptoms aggravated. Passive elevation of the knee from the bed, or attempt at producing ankle clonus threw, first the one, then both limbs into violent trembling. For a time crutches were used, but had to be abandoned. On February 1st, 1882, it was noticed by the patient when he tried to stand, that there was scarcely any feeling in the soles of the feet; the ground, which simply felt warm to the feet, seemed to be sinking under them. On test-

ing them, the prick of a pin was readily felt. Rigid flexion was noticed to come on for the first time on May 16th. It first occurred in the right lower limb, but in a few days affected the left also. The limbs could not then be extended passively. The spasm was not permanent; at times they could be extended voluntarily. On May 19th the urine was alkaline and purulent, and bed sores had formed over the sacrum and trochanters. A night's rest in a sitting posture gave a gangrenous look to the whole nates. However they recovered again. Wasting of the muscles at the front and back of the legs was noted then for the first time. On June 15th, patient complained of stiffness of the upper limbs. On examination they yielded very slowly to passive movement, both in flexion and extension, but there was no spasm that could not be overcome. There was no pain, tenderness, heightened reflex, or clonus in them.

The bed sores penetrated deeply to the bones through thick pads of fat. During the last week the temperature steadily rose, with remissions, accompanied by rigors and sweats, to 105-8° F. This was on July 12th: on the same day, the bed sore over the right trochanter bled freely. The next day the patient died. Temperature normal an hour before death.

Autopsy.—Body fat; lower limbs wasted. Serous cavities, heart, lungs, intestinal tract, liver spleen and kidneys normal. Rugæ of bladder congested, and trigone covered with mucus.

Brain and membranes, spinal canal and dura mater healthy.

The spinal cord presented marked gelatinous degeneration in the lateral columns which extended from the tip upwards into the upper portion of the dorsal region. The degeneration extended higher in the right side, and at a point above any visible sclerosis the column was congested, being in marked contrast to the rest of the white matter. In the lumbar enlargement the degeneration extended forwards into the anterior column and in front of this the white matter was greyer and firmer than normal. In one spot the anterior columns next the anterior median fissure were gelatinous. Above the lumbar enlargement the sclerosis did not invade the anterior columns to the same extent, and higher still the lateral columns were only in part affected. In the upper part of the dorsal region the gelatinous degeneration was visible as a thin band, just in front of the posterior cornu and parallel with it. After hardening in chromic acid the parts described above as sclerosed were stained brown, the tint varying with the degree of degeneration. Besides, the central grey matter was also stained in a similar manner in certain parts. In the mid-dorsal region and below this (except in the largest part of the lumbar enlargement), the grey matter was throughout darker than at the upper part of the cord. Above the mid-

dorsal region the brown staining invaded less and less of the grey matter, the anterior cornua being free from it, while the posterior half of the grey mass was stained. Higher still, at a point where the sclerosis in the lateral columns was shown by a thin wedge-shaped tract on the right side, and a thin band on the left, the grey matter was very little discoloured.

Microscopic examination showed the lateral columns and neighbouring part of the anterior to be nearly devoid of nerve tubes at the lower end of the cord, and the anterior columns to contain a considerable quantity of connective tissue between the nerve tubes. In the dorsal region the lateral columns contained some nerve tubes, which were found in increasing numbers as the examination was extended upwards. The most sclerosed part of the column seemed to be not directly in contact with the posterior cornu. In the mid-dorsal region the anterior columns were comparatively free from connective tissue. The large cells of the anterior cornua were much atrophied in the lower half of the cord, except in one section taken from the lumbar enlargement; in this the cells appeared normal in size, and number; but a little lower where the cells were in three pretty well defined groups, a small central group, a larger antero-lateral and a large postero-lateral group, the two first groups were markedly atrophied on the right side.

Remarks.—In 'The Localization of Cerebral and Spinal Diseases,'[1] Charcot, in attributing the symptoms of spasmodic tabes to primary sclerosis of the lateral columns, says "the hypothesis at the present time has received no verification."[2] Although in the above case the sclerosis extended beyond the lateral columns at the lower end of the cord, the symptoms, taken together with the extent and degree of sclerosis of these columns found post mortem, make it more than probable that this was a case of primary ascending sclerosis of the lateral columns.

[1] New Sydenham Society's Translation, p. 304.

[2] Dr. Dreschfeld has published a case since Charcot published this: *vide* British Med. Journ. 1881.

ELECTRO-THERAPEUTICAL NOTES.

BY WALTER G. SMITH, M.D. DUBLIN, F.K.Q.C.P.I.

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NOTWITHSTANDING the abundant evidence which has now accumulated in proof of the palliative and curative powers of electricity in the treatment of neuralgic affections, it must be allowed that its use is not yet so general as its undeniable claims to consideration justify. No consulting room can be said to be properly appointed that is not provided with a suitable induction apparatus, and a constant battery and accessories, *kept in good order*, and it is a sign of imperfect acquaintance with the teachings of modern therapeutics, if the medical practitioner neglect to avail himself of the resources which the legitimate use of electricity places at his command. I have no hesitation in expressing my opinion that in most cases of persistent or recurrent neuralgia, it is worth while to try the effects of a gentle galvanic or faradic current. When cautiously used, no ill-effects will ensue, and in many instances the success is so immediate and unquestionable as to be alike agreeable to the patient and gratifying to the physician.

Contrasting strongly with the important data to be gathered in affections of the motor apparatus, I am not aware of any observations tending to establish the diagnostic value of electricity in sensory affections, e.g. in distinguishing a central from a peripheral lesion; or, in framing a prognosis as to the mildness or gravity of a case.

In regard to the method of application of the current, it was for a time customary, and some writers still speak with confidence as to the relative merits of the so-called ascending or descending current, i.e. primary importance is attached to the direction in which the current is supposed to flow. This is a point which has occasioned much vain controversy and given rise to no little confusion of ideas, and to many idle and pseudo-scientific speculations. Clinical observers arrived at

discordant results, or came to the conclusion that it was immaterial whether the direct or inverse current were employed. But we have learned to regard the matter from another point of view, and now recognise the importance of studying the "polar" effects of the current, i.e. the local influence exercised immediately beneath, and in the neighbourhood of either electrode placed over the desired spot, the other electrode resting on an "indifferent" locality in the median plane of the body.

Electro-therapeutical science is indebted to the recent investigations of Erb, and of Waller and De Watteville, confirming and extending our knowledge of the polar effects of electric currents, which have done much to elucidate a difficult problem in physiology, and to dissipate a cloud of perplexities and apparent contradictions which obscured the rational application of electro-therapeutical methods. It should be clearly understood that the dispute as to the relative effects of the "polar" (uni-polar) and "direction" (bi-polar) methods is really a verbal one, and that there is no essential difference between them, both resting upon the same fundamental principle.

In the field of *diagnosis* of motor affections, we have gained a good deal in exactitude of knowledge, and it is to electro-physiology that we look for correct methods and ways of procedure, and for the destruction of erroneous views. But while we fully recognise that electro-therapy cannot dare to emancipate itself from electro-physiology on account of the practical insufficiency of existing physiological data, we must face the fact that a rational system of electro-therapeutics is still to be waited for, and we must be content for the nonce to let it root itself in the soil of clinical experience. A system of therapeutics based upon the theory of electrotonus, for example, rests upon an unsubstantial foundation.

That brilliant cures in many neuralgias can be obtained by electric currents employed in very different modes, and that in the same disease similar successes can be got by physiologically opposed methods is undeniable, and in illustration of this I will cite a personal observation of Rossbach. For many years Professor Rossbach had been a firm believer in the soothing action of the anode. He, unfortunately, became the subject of a very severe sciatica, and in the course of treatment of this affection by electricity, he was convinced, in the clearest way, that the pain was relieved, not only when the anode was placed upon the painful points, but even much more quickly and decidedly, when the kathode was placed upon the same points and voltaic alternatives employed. In another case, one of agonising facial neuralgia in an old

woman, he perseveringly applied the anode of the galvanic current for a long time without the slightest benefit, whereas the pain was relieved by the faradic brush in two short sittings. ('Lehrbuch der physikalischen Heilmethoden,' Dr. M. J. Rossbach.) Cf. abstracts of 17 cases in Erb's 'Elektrotherapie,' pp. 493–497. In conclusion I will adduce brief notes of some cases exemplifying the striking and permanent utility of electricity in controlling neuralgic conditions. Three of these cases occurred in the persons of medical men, two of whom wrote to me an account of the benefit they had experienced from the treatment.

Case 1.—Mr. J. C., aged 21 years, subject for several years in summer to attacks of neuralgia of the right side of the face. In June 1878, he was seized with severe pain over the right side of his face, especially about the upper and lower jaw. The pain came on in paroxysms 3 or 4 times a day, each paroxysm lasting about an hour. Exposure to a draught, or the act of drinking a cold liquid, would provoke an attack. The galvanic current (4–8 Leclanché cells) was applied for a few minutes; + pole over the painful spots, and − pole lower down and beneath the angle of the jaw. Immediate relief was obtained, and next day the current was again applied as some pain lingered about the orbital region. From the day following he was entirely free from pain, and has never, to my knowledge, had a relapse.

Case 2.—In March 1878, a young gentleman, Mr. B., consulted me for a violent attack of neuralgia in the right eye, of 14 days' standing. The pain shot through the ball of the eye constantly, causing lachrymation, and extended to the supra-orbital region.

He had taken quinine freely, and morphia had been injected subcutaneously over the eye, but without any benefit.

The continuous current was directed over the seat of pain, and, *after the first application*, the pain completely disappeared and did not return.

Case 3.—Dr. W., of middle age, subject since 19 years old to attacks of pain about the right hip and down the leg, subsequent to a fall. He has been laid up for 3 weeks at a time with sciatica, and ascribed his last attack to bathing. The current from 12 cells was applied; one electrode over the lumbar vertebræ, the other down the back of the thigh. Good effects were at once apparent, and the current was used some 6 or 7 times. He felt well and comfortable the whole day after the electrical treatment, was enabled to face cold wet

weather without fresh pain being provoked, and was so convinced of the beneficial effects of electricity in his own person, that he procured a Leclanché battery, and on future occasions was accustomed to check the first threatenings of return of the pain by a timely application of the current.

Case 4.—Reverend J. C. was on the point of starting for the S. of Ireland, when he was suddenly seized with agonising pain in the loins and hips. Hypodermic injections of morphia and other sedatives proved of little use. When I first saw him, at Dr. Head's request, he had been several days ill, was lying on the bed, sweating with pain, and the least stir caused such distress that he had not dared to undress himself, and every act of respiration sent a twinge of pain across him. He had not slept for some nights. The effects of the continuous current applied to the painful parts was very striking. After a few minutes he ventured to turn in bed, was then able to undress and lie comfortably in bed, and in 3 or 4 days he left for Cork, and has since had no return of the pain.

Reviews and Notices of Books.

History of the Criminal Law of England.—By Sir JAMES FITZJAMES STEPHEN, K.C.S.I., D.C.L., a Judge of the High Court of Justice, Queen's Bench Division, London. Macmillan, 1883.

THIS important work contains a great chapter of especial interest to medical men, on "The Relation of Madness to Crime," so different in tone and quality to anything which has before been written by anyone of the author's profession that it stands, and cannot fail to remain, as a mindmark from which all further advance in the solution of one of the most difficult problems of legal and medical and social science must be measured. "The plea of insanity is unquestionably the most difficult corner of the Criminal Law" once said to us a great judge, than whom no one who ever thought on the subject was more competent to form an opinion; and a difficult corner we fear it will ever remain, from the inherent obscurity and intricacy of the questions involved. And this new contribution to its literature is far from clearing away the difficulty, notwithstanding the spirit of wisdom and justice, and the great knowledge and authority with which it is written. Indeed, so far as it widens the legal view of irresponsibility on the ground of insanity, it certainly will not tend to lighten the burthen of responsibility hitherto borne by our Courts in these arduous inquiries. For, so long as our judges are content to lay down the law in the simple and concise formula that a man who intentionally commits homicide, knowing the nature and quality of the act, and that it is wrong, is punishable as a murderer, the law is not difficult to be enunciated; it is only difficult to apply, because it does not conform to the facts of human existence. But if the control of conduct, as influenced by disease, be admitted, as eventually it must be, into the consideration of guilt or innocence, an element of even greater intricacy than that of knowledge of the nature and quality of the act, or that it was wrong, will be added to the law of insanity, and although justice will be rendered more secure, its determination will not be facilitated.

There would seem to be three sufficiently distinct modes of considering the irresponsibility of homicides, in whose defence the plea of insanity is advanced. First, there is the old customary manner of stating the law, which requires the defence to prove inability (arising from insanity) to know the nature and quality of

the act, or that it was wrong; and there is some doubt as to whether the word "wrong" means legally wrong only, or that it may mean morally wrong. Secondly, there is the proposed modification of the law, inability to control conduct caused by disease; and, Thirdly, there is what may be called the physiological, in contradistinction to the two first, which may be considered the legal modes of considering this question. The physiological mode we should say is to add to the considerations as capable of determining the question at issue, the state of the man's mind with regard to the particular act, as to whether he knew its nature and quality, or whether it was wrong legally or morally, or whether he was in fact and deed capable of resisting the desire to commit it, the consideration whether or not the accused was or was not at the time of the act suffering from disease affecting his mental functions so as to render him generally incapable of reasoning, feeling and acting like other men. And it is not presumptuous to affirm that this is the only mode in which the mental results of disease can be considered exhaustively. To a certain extent it includes both of the legal modes, but it is by no means merely commensurate with them. For while no insane man really knows the nature and quality of any of his actions, in the full sense of the knowledge of a rational man, and no insane man recognizes as a rational man does the full extent of the wrong he commits by a criminal act; at the same time, a large proportion of the insane do know to a great extent what they are doing and whether it is wrong, and also they can very frequently resist very strong temptations, whether they be of a natural or morbid kind. There is no mental function which is not liable to be affected by an attack of insanity, neither is there any mental function which is usually quite suspended or destroyed by such an attack of insanity as is likely to leave a man capable of committing an offence and likely to be punished for it. Moreover, there are mental functions affected, or disabling mental states produced by disease which can scarcely be connoted by any meaning of the term "knowledge." It would seem, therefore, that there is no mental test which lawyers or legislators can adopt which is not liable to fail if it be made narrow, definite and absolute. Such tests, for instance, as does not know, cannot help it, cannot control his conduct, when, in fact, he does know to a great extent, can help it, and can control his conduct, if the inducement to do so be sufficiently strong and urgent.

The author declares, and there can be no doubt that he is right, that "the question, What are the mental elements of responsibility? is and must be a legal question. It cannot be anything else, for the meaning of responsibility is liability to punishment; and if criminal law does not determine who are to be punished under given circumstances, it determines nothing."

But the question before a Criminal Court, is not what are the elements of responsibility, but whether these elements do or do not exist in the individual accused of an offence. And when it is affirmed that these elements are absent, in consequence of bodily

disease affecting the prisoner's mind, the question becomes not a legal but a medical one; and this the author to a certain extent acknowledges. He says, "I think it is the province of the medical men to state for the information of the Court such facts as experience has taught them bearing upon the question whether any given form of madness affects, and in what manner, and to what extent it affects, either of these elements of responsibility, and I see no reason why, under the law as it stands, this division of labour should not be fully carried out."

But this division of labour would render medical evidence general and impersonal, and of less real value in determining innocence or guilt than the opinion of any common man, a ploughman or a sailor, who had known the accused and could testify as to what he thought of him. The author must therefore have intended, in the division of labour which he approves, to indicate the evidence which a physician may be permitted to give who has made no personal observation of the individual accused. This kind of evidence is now very properly restricted in criminal trials to the generalities with which it deals, which have a certain value, but which are not comparable to the evidence of detailed personal observations made by competent medical men, which ought always to be available in so grave an inquiry as the responsibility of a murderer defended on the plea of insanity.

We take it, therefore, that the division of labour approved of by the author relates to the division of labour in the formation of opinion, more or less authoritative, in generalities. Firstly, there is the almost absolutely authoritative opinion of the judge as to the mental elements of responsibility; and, secondly, the opinion of the medical authority, as to whether a certain form of madness does or does not include those elements. For instance, the judge, with the authority of his high office, gives the jury what may be called the order that they must only consider as the mental elements of responsibility the knowledge of the nature and quality of the act and that it was wrong. And the physician who has not examined or personally known the accused, may be asked whether a state of mental depression without insane delusions is inconsistent with those mental elements, and it would not be permissible for the physician in such a case to state his opinion that there were other elements of responsibility, than those laid down by the judge.

Unfortunately the opinions of medical men on responsibility vary widely, according to their character, education and experience, but the law of the matter, that is to say the mental elements of responsibility, ought to be absolute and definite. This, however, would seem to be by no means the case, and the uncertainty seems to be fully admitted by the author, who says:—

"I believe that by the *existing* law of England those elements (*i.e.* the mental elements of responsibility) so far as madness is concerned, are knowledge that an act is wrong and power to abstain from doing it. I think it is the province of the judges to declare and explain this to the jury." "If I am wrong in thinking that the power to abstain from a given act is an element of respon-

sibility for it, the duty of the judge is to tell the jury that such is the law, and to exclude from the consideration of the jury as being irrelevant, all evidence leading to show that the accused person was deprived by disease of control over his actions."

It would appear from this that Sir James Stephen, a judge of the High Court of Justice, is not certain as to what is the law of England in this important matter, and those who have studied his fair-minded and candid pages will be the less surprised when they have observed other judges in recent trials laying down the law of England as he would not have done. On a quite recent occasion, for instance, Mr. Justice Day, in the trial of William Gouldstone, on the 14th September last, for the murder of his five children at Walthamstow, is reported in the *Times* to have charged the jury thus: "He told them as a matter of law, that if the prisoner, when he killed the children, knew the nature and the quality of the act he was committing, and knew that he was doing wrong, then he was guilty of wilful murder. The nature and quality of the act meant that the man knew what it was that he was doing, that is to say that he knew he was killing a fellow-creature. He repeated that if a man killed a fellow-creature, knowing at the time he was doing wrong, then he was guilty of wilful murder. The only question therefore for them to determine was whether the prisoner knew the nature and quality of the act he was committing, and whether he knew it was wrong." In this enunciation of the law on this most solemn occasion there was nothing said of that mental element of responsibility which consists in the power to abstain from a given act, and therefore either the "existing law of England" has been too widely stated in the author's pages, or it was most inadequately stated in Mr. Justice Day's charge.

That Mr. Justice Day's law was held by the judges generally to be the existing law of England up to a comparatively recent date cannot be doubted, for Lord Chief Justice Cockburn, in his Memorandum to the Select Committee on Sir James Stephen's Homicide Bill, in 1874, remarks: "As the law as expounded by the judges in the House of Lords now stands, it is only when mental disease produces incapacity to distinguish between right and wrong that immunity from the penal consequences of crime is admitted. The present Bill introduces a new element, the absence of the power of self-control. I concur most cordially in the proposed alteration of the law."

The law, however, has not been altered by statute, and it is an interesting and important question in what manner and at what time it has been or may be altered by the individual or collective action of the judges. The alteration will certainly meet with the concurrence and approval of the medical profession.

It is quite a novelty to find a work of this nature, scope, and authorship, founded and argued upon a large appreciation of medical opinion; although with regard to such opinion and authority the author is evidently, and perhaps necessarily, somewhat in the same position in which he describes medical men to be in with regard to the law, when he says:

"I must say that the provisions of the existing law have, as it seems to me, been generally, though perhaps not unnaturally, misunderstood by medical men, who cannot be expected either to appreciate the different degrees of authority to be ascribed to different judicial declarations of the law, or to understand the rules for their interpretation, or to recognize the limitations under which they are made, or to appreciate the fact that when made they cannot be altered at will. Moreover, to read judicial decisions correctly is an art in itself, to be acquired only by long professional practice, nor can any one even begin to do so before he has familiarised himself with several rules well known to lawyers, but, in my experience, altogether unknown to medical men."

Although this may not be quite true in its *vice versâ* application to the interpretation by lawyers of that medical science which we are so often blamed for rendering needlessly recondite and technical, still it is so far true that the balance of medical opinion can scarcely be fairly held by any hand which has not been trained in the gymnasia of physiological study. And this is peculiarly the case in a department of medical science so new and developing, and yet so incomplete, as that of mental pathology. But while the pages of our author afford ample evidence that the legal mind is capable of grappling with the most intricate and difficult medical questions, so far as his professional duties require them to be understood, he would, we are sure, admit that medical men who gave their minds to it, might attain to the power of correctly reading judicial decisions. An example of the author's professional view of insanity as distinguished from the physiological view, is seen in the tendency which he nowhere exactly expresses, but which makes itself felt throughout his pages, to consider insanity as one condition the development of which is explicable according to Griesinger, whom he largely quotes, on the Guislain theory of phrenalgia; and another example in his acceptance of the theory of moral insanity. Our limits do not permit more than the expression of what we believe to be the true physiological view, that insanity is but a symptom of a great number of bodily diseases, and that the theory of moral insanity promulgated on such insufficient grounds by Pritchard and Ray, is to be relegated to the long list of exploded errors which occur in the gradual development of a difficult science. But considering the yet existing imperfection of this science, and the bias which the legal mind almost necessarily has, to consider exclusively what are called the mental elements, the ampleness of the conclusion arrived at by the author is, it must be admitted, far in advance of all former legal authorities. This conclusion is:

"As to the verdict of not guilty on the ground of insanity, the foregoing observations show in what cases it might in my opinion be returned; that is to say, in those cases in which it is proved that the power of self-control, in respect of the particular act, is so much weakened that it may be regarded as practically destroyed, either by general weakening of the mental powers, or by morbid excitements, or by delusions which throw the whole

mind into disorder, or which are evidence that it has been thrown into disorder by diseases of which they are symptoms, or by impulses which really are irresistible and not merely unresisted."

This summing up of the mental elements of irresponsibility appears to us admirable, although we can scarcely admit that even yet it is perfect. Perhaps, for instance, the addition of morbid defects to morbid excitements would widen its scope and strengthen its significance. It is however the most comprehensive condensation of what ought to be the law which we have ever met with by any authority, legal or medical, and it is especially to be noted that its whole structure is based upon the foundation of the loss of self-control, thus differing in an essential respect from the author's former attempt to formulate a reasonable and satisfactory dictum.

For in Sir James Stephen's Homicide Bill, which was the subject of the Select Committee in 1874, he proposed to codify the law in the following manner:

"24. Homicide is not criminal if the person by whom it is committed is at the time when he commits it prevented by any disease affecting his mind. *A.* from knowing the nature of the act done by him. *B.* from knowing that it is forbidden by law. *C.* from knowing that it is morally wrong; or, *D.* from controlling his own conduct. But homicide is criminal, although the mind of the person committing it is affected by disease, if such disease does not in fact produce some of the effects aforesaid in reference to the act by which death is caused, or if the inability to control his conduct is not produced exclusively by such disease. If a person is proved to have been labouring under any insane delusion at the time when he committed homicide, it shall be presumed, unless the contrary appears, or is proved, that he did not possess the degree of knowledge, or self-control hereinbefore specified."

In our Lumleian Lectures of 1878, on Insanity in its Legal Relations, we recognized the above "as the only earnest and serious attempt which has been made to amend this stereotyped fragment of the common law," but we proceeded to criticise it as follows:

"It is evident that the pith and substance of this scheme of law is comprised in its fourth section, which, like Aaron's rod, turned into a serpent, swallows up all the others. If we complete each sentence my meaning will be apparent. The full expression of exemption ought clearly to be as follows. Homicide is not criminal if the person by whom it is committed is, at the time when he commits it, prevented by any disease affecting his mind from knowing the nature of the act committed by him and therefore from controlling his own conduct. Between the not knowing and the doing there is a great gulf fixed, which can only be bridged by words conveying the idea of motive; the words used 'prevention of control' being most convenient.

"From this consideration it appears to me demonstrable, that clause D of the proposed code virtually includes all the others, and is sufficient in itself. 'Homicide is not criminal if the person by

whom it is committed is, at the time when he commits it, prevented by any disease affecting his mind from controlling his own conduct.' This could scarcely need any further development in terms, if it were only wisely acted upon in the full spirit of Lord Justice Blackburn's dictum, that, in every individual case, you must look at the circumstances, and do the best you can to say whether it was the disease of the mind, which was the cause of the crime, or the party's criminal will."

That this loss of self-control is not merely to be considered as an addition to the anciently recognised elements, but that it is the one essential though secondary element of irresponsibility, we pointed out in another of our Lumleian Lectures, as follows:

"There is one attribute of conduct produced by disease which is sufficiently simple to be intelligible by men of plain common sense, sufficiently constant not to cheat us with false hopes of guidance, and directly applicable to the end in view, and this is the uncontrollable character of conduct resulting from disease of the brain. Surely it must be that this quality of conduct resulting from disease has always been at the root of the law, although even up to the present time it has never been expressed in the law; for if this quality be abstracted, what reason would remain for excusing a man from punishment, on account of ignorance or unreason, even if he be insane? If a madman could be made to control his conduct like other men, we can scarcely believe that any tenderness for his misfortune would be able to overrule the obvious expediency of compelling him to do so; while in his deprivation of self-control, and his insubjection to imposed control, we have a test of his madness and a practical reason for his irresponsibility."

We insist greatly upon the importance of assigning to loss of self-control from mental disease its proper place and function as the secondary but immediate and exciting cause of the homicidal act, while ignorance and delusion have assigned to them their proper place and function as remote causes, because it appears to us that to admit loss of self-control, without reference to other mental states intermediate between the diseased organism and the action as a ground for irresponsibility, would be inconsistent with psychological truth and dangerous to the administration of justice, by sanctioning the widest theory of insanity of the emotions without any trace of intellectual aberration or defect. It is therefore with great satisfaction that we note the decided change which has taken place in this respect in the author's proposed enunciation of the law since he drew his Homicide Bill some ten years ago. The principle being admitted, that loss of self-control caused by disease primarily affecting the intellectual functions is the one constant element of the irresponsibility of the insane, the most convenient dictum in which it can be expressed will no doubt be eventually eliminated, if it has not already been attained by the author in the passage last quoted from his most recent work. Perhaps it may be that the best formula of words, or even the truest ideas, have not yet been quite reached. Perhaps other modes of mental diseases in addition to weakness, excitement, and delusion, may be recognised;

and very likely the uncomplicated existence of "impulses which really are irresistible and not merely unresisted" will be found an idea too vague and uncertain to be admitted into the administration of the criminal law.

We have to observe another important improvement in the author's present suggestion of the law as it ought to be, in his omission of all circumstances which render "a homicide criminal, although the mind of the person committing it is affected by disease." Some of these, as stated in the Homicide Bill, are tautological, but the provision which affirms that homicide by a person affected by mental decease is still criminal, "if the inability to control his conduct is not produced *exclusively* by such disease," appears to us quite unworkable. What action of any lunatic can ever be predicated as produced exclusively by disease? Clearly none. The knowledge which a man has acquired before he has become insane goes largely to the direction of his conduct, for, as far as knowledge is concerned, the maddest action is more the result of sanity than of insanity, if it be possible, even theoretically, to discriminate different portions of a man's mind at the same moment into the two states. And, in practice, how would it be possible to say of an insane drunkard that so much of his conduct was due to madness, and so much to drunkenness? Take as an example the lunatic who shot a provision dealer at Holloway, and was found guilty, we believe, because it was proved that he was drunk at the time. It was, however, pointed out to the Home Secretary that this man had attempted to gain access to his victim, for the purpose of homicide, three or four hours before he actually did commit it, and when he was quite sober, though at both periods he was under the influence of insane delusion; and the man was very properly respited. Such cases are not rare, nor the still more difficult ones in which an obviously insane person is influenced to a criminal act by a natural but malicious motive; a motive, however, which would not have led to its actual result but for the mental disease; and it must be impossible for human intelligence in such a case justly to apportion the blame and the punishment. We are glad, therefore, to find that the author in his most recent dictum omits this impracticable condition of irresponsibility. Another somewhat similar question discussed by the author, although wisely omitted from his dictum as to what the law ought to be, is to what extent any lunatic ought to be held responsible for his madness, and whether the disease which causes the mental elements of irresponsibility ought to be defined as disease not occasioned by his own default. He states his grounds for this suggested alteration of the law as follows:

"In reading medical works, the connection between insanity and every kind of repulsive vice is made so clear that it seems more natural to ask whether in many cases insanity is not rather a crime in itself than an excuse for the crimes which it causes. A man cannot help an accidental blow on the head; but he can avoid habitual indulgence in disgusting vices, and these are a commoner cause of madness than accidents. He cannot avoid the misfortune

of being descended from insane or diseased parents; but even if he has that misfortune, he ought to be aware of it, and to take proper precautions against the effects which it may be expected to produce. We do not recognise the grossest ignorance, the most wretched education, the most constant involuntary association with criminals, as an excuse for crime; though in many cases—I think in a smaller proportion of cases than is commonly supposed—they explain the fact that crimes are committed. This should lead to strictness in admitting insanity as being in doubtful cases any excuse at all for crime, or any reason for mitigating the punishment due for it. It is upon this ground that I think that the general rule that a person should not be liable to be punished for any act done when he is deprived by disease of the power of controlling his conduct should be qualified by the words "unless the absence of the power of control has been caused by his own default."

This proposal is simply impracticable, for with the exception of the few cases of traumatic insanity referred to, are not most cases of insanity more or less caused by the patient's own default, in the author's sense of default? that is to say, that the person liable to its development ought to know his danger and to take proper precautions to avert it. When it is remembered that the disgusting vices which are a common cause of insanity are themselves very frequently the results of a lower degree or less developed form of mental disease; and when it is remembered that hereditary insanity is oftentimes a gradual development of obscure conditions from infancy upwards, it may safely be affirmed that it is beyond the reach of the highest human wisdom, and certainly far away beyond the rough and ready methods of our courts of criminal law, to distinguish how much of a man's madness is the result of his own default. And if it were the result of his own default, ought he not in justice to be punished for his madness which by the proposition he ought to have foreseen and avoided, and not for his criminal offence, which, theoretically, has been caused by the default of others in not having recognised the man's madness and taken due precautions against its results? And is not madness itself a sufficient punishment for the offence of neglecting one's health?

There are several other questions mooted and argued with vigorous common-sense, cleansed from all sentimentality, which seem to invite a reply, in which we should greatly like to see what could be said from the medical point of view, if our limits would permit. But we would conclude by strongly recommending Sir James Stephen's pages, not to the perusal merely, but to the diligent study of any medical man who may wish to obtain some mastery of one of the most arduous duties to which the incidents of his professional life render him liable. They will not, it is true, give him much knowledge of mental disease, but they will enable him to arrange such knowledge as he may otherwise have acquired, in such manner as to make it available for the most grave and special purpose of giving evidence with understanding. They are bound also to have an important effect upon future legislation, or, failing that, upon the unwritten law of the land. They cannot

be the last words on the subject, for they are too suggestive to be accepted as a settlement of many of the questions which they argue, but they very evidently constitute a new starting-point for anything of importance which can be said on the subject in the future.

JOHN CHARLES BUCKNILL.

Lehrbuch der Gehirnkrankheiten für Aerzte und Studirende.—Von Dr. C. WERNICKE. 3 vols. Fischer; Kassel, 1881–1883.

THE work before us is an exceedingly important one, and demands more than usual consideration at the hands of a critic. It aims at fulfilling the double function of an extended treatise on the diseases of the brain for the use of physicians, and a handbook for students. This two-fold object is endeavoured to be attained by the use of large and small type. The leading features of the anatomy, and the fundamental principles of the physiology of the brain, as well as the systematic description of the clinical phenomena, are printed in large type; whilst the details of the anatomy and the debateable questions of the physiology of the brain, as well as the reports of all the illustrative clinical cases, are printed in small type.

The work, so far as yet published, consists of two volumes of somewhat unequal size. The first volume is divided into two parts: in the first part, which extends over 272 pages, a full and elaborate account is given of the anatomy and physiology of the brain, and in the second part, which occupies the remaining 100 pages of the volume, the individual symptoms of cerebral disease are described and explained. The second volume, which consists of 251 pages, is occupied with the consideration of the general pathology of focal diseases, but the only diseases of this class which the author has discussed up to the present are hemorrhage and softening. It will thus be seen that this work is a mere fragment of what we may expect it to attain. The wide subject of intracranial tumours has yet to be dealt with, and we may rest assured that the author will not decline the consideration of the diffused inflammations of the brain and of its membranes, or such affections as shock, concussion, neurasthenia cerebralis, epilepsy, and hysteria.

The first part of the first volume, which treats of the anatomy and physiology of the brain, is by far the most important section of the work. This part occupies rather less than one half, and considerably more than one third of the whole book, and is illustrated by 96 woodcuts. Of the woodcuts, 21 are purely diagrammatic, and illustrate the text which is printed in large type, and which is intended for the use of students. These diagrams are admirably conceived, and they render the course of the various communicating fibres and conducting paths as clear as the complicated nature of the subject will permit. Of the remaining figures a few are borrowed from Flechsig, Charcot, and Munk, but by far the greater number are original, and are drawn from nature. Everyone accustomed to

examine horizontal sections of the hemispheres, the crura, pons, and medulla oblongata will at once recognise the extreme fidelity of these figures; nothing can exceed their delicacy of execution, and they do equal credit to the artist, the engraver, and the printer. The lines are so delicate, and for the most part so well defined, that the figures look more like steel engravings than woodcuts. In a few of these figures, however, a somewhat bolder treatment would have been desirable, the representation of a section of the lower end of the medulla oblongata [Fig. 69] being, for instance, so indefinite that it is difficult to trace any structure whatever.

The account which Wernicke gives of the anatomy of the brain differs in some noteworthy respects from that of other recent authors, while his own original contributions to cerebral anatomy are by no means few or unimportant. In describing the surface of the frontal lobe the author, following the earlier anatomists, such as Leuret, numbers the convolutions of the upper and outer surface of the lobe from below upwards, instead of from above downwards as is done by Wagner and Ecker. If the reader fail to remember this, he will be startled to find it asserted that motor aphasia is caused by lesion of the posterior extremity of the *first* left frontal convolution.

The longitudinal or collateral system of fibres are usually said to consist of (1) arcuate fibres or fibræ propriæ, (2) fibres of the gyrus fornicatus, (3) the fasciculus uncinatus, and (4) the inferior longitudinal fasciculus.

In addition to these, Wernicke describes for the first time a large bundle of fibres which, from the course they pursue, he names the *perpendicular occipital fasciculus;* these fibres connect the inferior parietal lobule and the fusiform lobule of the temporo-sphenoidal lobe. Nothing beyond conjecture can as yet be said with regard to the functions of this fasciculus. The author asserts that neither the caudate nucleus nor the external division of the lenticular nucleus is directly connected with the cortex by radiating fibres. He believes that these masses of grey matter must be regarded as independent ganglia, like the grey matter of the cortex itself. The first and second divisions of the lenticular nucleus he regards as ganglia of interruption, which connect the caudate nucleus and the third division of the lenticular nucleus with the central grey tube.

The opinion of Broadbent, Charcot, Flechsig and others that the fibres of the pyramidal tract pass uninterruptedly through the internal capsule or crusta is confirmed by Wernicke. The most noteworthy contribution which he has made to the elucidation of the constitution of the internal capsule, is his statement that those fibres of the corpus callosum, which form the anterior wall of the anterior horn of the lateral ventricle, wind backwards along the external border of the caudate nucleus to mingle with the descending fibres of the anterior segment of the internal capsule. The fibres of the pyramidal tract are said by Wernicke to be everywhere interrupted in the pons by grey matter, but we do not share this opinion. An attentive examination of sections,

especially from the embryo, and from cases of descending degeneration, will, in our opinion, show that, although the longitudinal bundles of the pons are often surrounded by grey matter, yet the fibres do not form connections with the ganglion cells of this substance.

The connection between the basal ganglia and the various tracts and ganglia of the crura cerebri are very carefully described, and illustrated, the diagram [Fig. XII.] which explains the connections between the different parts of the optic thalamus and the tegmentum being particularly instructive. The author has traced connecting fibres between the optic thalamus and the red nucleus of the tegmentum. But a case of descending degeneration secondary to a focus of softening in the pulvinar of the optic thalamus has recently been reported by Mendel,[1] which seems to show that these fibres pass along the inner margin of the red nucleus, and, then cross over to reach the superior peduncle of the cerebellum of the opposite side, where they could be followed as far as the corpus dentatum of the cerebellum. Connecting fibres between the optic thalamus and the fillet have been discovered by Wernicke. He also states that the innermost fasciculus of the fillet is derived from a bundle of fibres which is situated on the internal border of the crusta; this fasciculus does not reach the medulla oblongata, but terminates in the pon in grey matter.

The tract of fibres which lies close to the nucleus of origin of the third nerve, near the floor of the aqueduct of Sylvius, named the *posterior longitudinal fasciculus*, is said by Wernicke to be connected with the first division of the lenticular nucleus by means of the fibres which form the anterior portion of the collar of the crus. The posterior longitudinal fasciculus maintains throughout the whole length of the crus, pons, and medulla oblongata a close connection with the nuclei of origin and roots of the motor nerves, and it is continued downwards in the spinal cord as the inner division of the anterior root-zone. If this fasciculus is, as Wernicke asserts, connected with the lenticular nucleus, and not with the optic thalamus, as has hitherto been supposed, an additional argument is afforded in favour of the opinion that it is endowed with motor functions.

The fibres of the internal bundle of the optic tract are said by Wernicke to turn upwards in the external part of the crusta to reach the posterior portion of the internal capsule, and without forming any connections with either of the geniculate bodies or with the anterior tubercle of the corpora quadrigemina. Other noteworthy discoveries made by the author are that the fibres of the posterior bundle of the anterior commissure of the third ventricle terminate in the cortex of the temporo-sphenoidal lobe, and that the lenticular nucleus is connected by fibres with Luys' disc and with the locus niger.

These, then, are the chief contributions which Wernicke has

[1] Mendel, "Secundäre Degeneration im Bindearm;" 'Neurologisches Centralblatt,' Bd. 1, 1882, p. 24.

made to the anatomy of the brain, and it will be seen that they are both numerous and important. No part of the anatomy of the brain, however trivial it may appear, is without its value to the physician who knows how to decipher its meaning, and the man who possesses the most accurate knowledge of the structure of the nervous system in its minutest details, is likely to be the best clinical observer, as well as the best interpreter of what he does observe. Dr. Wernicke has now placed within our reach the fullest and best account of the structure of the brain, for clinical purposes, which has hitherto appeared in any language, and for doing this, special workers in the field of neurology owe him a deep debt of gratitude, which they will be forward to acknowledge.

The physiological part of the first section of the book, does not probably stand out so pre-eminently for its excellence as the anatomical part, but the account which the author gives of the experimental investigations of Fritsch and Hitzig, and of those of Munk, is good, and sufficiently full to form a ground-work for the interpretation of the clinical phenomena. The author also makes good use of the phenomena of the different forms of aphasia, which he divides into motor, conductive, sensory, and total aphasia, for the elucidation of the functions of the brain. A very useful account is likewise given of the course of the motor and sensory paths, which connect the cortex of the brain with the periphery. He adopts the views of Flechsig and Charcot in his description of the course of the motor conducting paths, while his discussion of the different routes which the sensory conducting tracts may possibly pursue, in passing from the spinal cord to the internal capsule, will be found to be exceedingly instructive.

Want of space forbids us to enter upon a detailed examination of the clinical part of the work. We have already seen that only a very small proportion of the diseases of the brain have as yet been described by the author, and we must give it as our opinion that the clinical portion, although full and good, hardly equals in excellence the anatomical and physiological section. It may be noticed in passing, that the author assents to the doctrine that epileptic convulsions are caused by irritative lesions of the cortex of the brain, and not by lesions of the medulla oblongata. It need scarcely be added that the theory of cortical discharge first enunciated by Hughlings-Jackson, and subsequently confirmed by the experiments of Ferrier, has for a long time been adopted by neurologists in this country.

We shall now point out what we conceive to be a few of the shortcomings of this work. This part of our task is undertaken in no carping spirit, and with no desire to detract from the great value of Dr. Wernicke's labours. We offer our remarks, indeed, with great diffidence, and with the full consciousness that the author may be, as so often happens, wholly in the right, and his critic wholly in the wrong. The first objection which we shall urge is, that there is an absence throughout the book of a fertile principle, like the law of evolution, and its converse the law of dissolution, which ought to run like a warp through the

entire tissue of facts, giving strength to the texture and unity to the design of the work. This defect leads to a want of unity in the different parts, and the author has been unable to utilise his very excellent though brief account of the development of the brain towards the elucidation of the phenomena of disease. Embryology, which ought to afford the best clue for unravelling the mysteries of disease, is, in Wernicke's hands, almost unused and useless. The author, following Meynert, begins his description of the brain with the cortex, and thence passes downwards by successive horizontal sections to end with the description of the junction of the medulla oblongata with the spinal cord. Much may be said in favour of this mode of procedure, but we believe it to be greatly inferior to the reverse method, which begins with the upper end of the spinal cord and ends with the cortex. The method adopted by Wernicke begins with the most complicated part of the brain, and ends with the simplest, but it would surely be wise to begin, after nature's order, with the simple and to proceed gradually to unfold the complex. It is manifest that the simplest portion of the brain is to be found in the embryological tracts of the spinal cord at their junction with the medulla oblongata, and consequently the best method for unravelling the complex structure of the organ, would be to follow these tracts to their various terminations in the medulla oblongata, pons, and crus cerebri, and then to trace their connections with the higher ganglia, and the connections of those ganglia with still higher ganglia, until ultimately the cortex is reached.

It is to be regretted that Wernicke has limited himself almost entirely to horizontal sections, and the introduction of Pitres' vertical sections of the cerebral hemispheres into this work would, in our opinion, be a great improvement.

In the clinical part the author discusses in full the pathology of focal diseases, first when the lesion is a hemorrhage and second when it is a softening. According to this method of procedure a great deal of what is said with regard to what may be called the localising symptoms in the section on hemorrhage has to be repeated in the section on softening, and will have to be repeated a third time when the author comes to deal with the subject of intracranial tumours. We believe that it would not only be an economy of space, but that it would also conduce much to clearness of treatment, were the author to discuss at first the general symptoms of hemorrhage, softening, and tumours separately, and subsequently describe the special or localising symptoms of all these lesions in one section. Many of the symptoms of disease of the lenticular nucleus, for instance, will be the same whether the lesion be hemorrhage, softening, or tumour, and nothing but confusion can arise by describing these symptoms separately for each lesion. Probably the most serious blot on the whole work, is to be found in the fact that the description of the blood vessels of the brain, according to the researches of Heubner and Duret, is to be found as a kind of appendix to the second volume, and consequently after instead of before the consideration of the

subjects of hemorrhage and softening. The absence of illustrative diagrams of the arteries of the brain is also a serious drawback.

We observe with pain that, although a somewhat grudging acknowledgment is made of the value of Ferrier's experimental determination of the cortical motor centres of the monkey, yet what we conceive to be his more important investigations of the sensory cortical centres is passed over with significant silence. It is possible that the experiments of Ferrier and Yeo on the occipital lobes may not have been published before Wernicke's work had passed through the press, but this cannot, of course, be said of Ferrier's earlier experiments for the determination of the sensory centres. We are aware of all that can be said against the validity of these experiments, but the facts still stand that Ferrier was the first physiologist who broke ground in this direction, and that the conclusions at which he arrived, from the admittedly imperfect data at his command, are marvellously accurate, even when regarded from the standpoint of our present more advanced knowledge. We know that there are several reasons why Ferrier's work should be less appreciated at Berlin than elsewhere, and all we can say is, that after giving full weight to these reasons, and after making a strenuous endeavour to place ourselves in the attitude of a Berlin physiologist with relation to the facts, we are totally unable to sympathise with the spirit which has dictated this want of generosity towards Ferrier and his work.

In bringing this review to a close, we may be allowed to express the opinion, that even after giving due consideration to every fault and shortcoming which the most captious critic can point out, the work before us must still be regarded as an extremely valuable one, and a noble contribution to neurology. These volumes are, indeed, well worthy of the great school of medicine from which they have emanated; they cannot fail to enhance the already high scientific reputation of their distinguished author, and we feel assured that for many years to come they will not be found far from the elbow of all who have to lecture upon, or make a special study of the diseases of the nervous system.

Since the foregoing remarks were written the third volume, or rather the second part of the second volume, of Dr. Wernicke's work has come to hand. It deals with the subjects of intracranial tumours, abscess of the brain, diffused multiple sclerosis of the brain (chronic polyencephalitis), the various forms of cerebral meningitis, idiopathic hydrocephalus internus, thrombosis and phlebitis of the cerebral sinuses, and progessive paralysis of the insane. This portion of the work extends over 300 pages, and the various subjects comprised in it are discussed in a very elaborate and satisfactory manner, each disease being fully illustrated by valuable records of clinical cases. So far as we can judge from a necessarily cursory examination, the new volume attains a higher level of excellence than has been reached in the clinical portion of the earlier volumes.

JAMES ROSS.

The Brain, considered Anatomically, Physiologically, and Philosophically.—By EMANUEL SWEDENBORG, Edited, Translated and Annotated by R. L. TAFEL, A.M., M.D., Ph.D. James Speirs, 36 Bloomsbury Street, London.

THIS is without doubt one of the most remarkable books we have seen, and perhaps to a great extent, on that very account, it is a very difficult task to criticise or review it. The distinguished author lives in history more as the founder of a form of religious thought than as a scientific man, and yet it is a fact that he was eminent as a mathematician, as an engineer, and as a mineralogist and geologist. Still it is a new experience to find him figuring as the writer of a work on the human Brain, and still more curious to discover that, although that work has lain by for nearly a century and a half, it is now not only not valueless, but it appears actually to have anticipated some of the most modern discoveries in the region of its subject-matter. Only the first volume of this remarkable work is before us, but we are bound to say that the prevailing feeling forced on our minds during its perusal has been one of surprise at the great power and profound philosophical insight of the writer, as point after point of his argument has been brought into view. No doubt, to Swedenborg, this work had mainly a theological interest. He was over fifty years of age when he wrote it, but it was about this time in his life that he directed his energies mainly into those lines from which arise his claims to live in the temple of fame, and the remaining thirty years of his life were employed in elaborating the views he had already formed. This fact undoubtedly makes it more difficult to gauge the value of the work, because for us it has only a scientific interest, and it is therefore necessary to unravel, and, so far as possible, to confine to its rigidly scientific bearing the phraseology, now metaphysical, now ontological, now theological, of which the author makes use. And indeed we may say that if it had not been that attention was arrested and interest enchained by finding so many anticipations of scientific discoveries by as much as 120 or 130 years, we should have been tempted to throw aside the book as beyond our province, if not hopelessly unintelligible. For it is certainly difficult to deal scientifically with such expressions as "essences of a superior and inferior kind," "the ideal of the universe which is below," "the supreme in every system of the soul," and such as "series determined into higher and lower spheres," with other like phrases which occur from time to time. In order, however, once for all, and so that repetition may be avoided—in order to make as it were the transition from Swedenborg's ontology to the scientific sphere, in which alone for the purposes of this review we are interested, it seems necessary in the briefest manner, to make some remarks which shall show at once the nexus and the separation between the two. Swedenborg assumes all through his writing the existence of the soul, which, according to him, has its seat in

the minute parts (each as it were a smallest brain with its own vessel, its own grey substance, and its own fine membrane) of the cortical substance; and he always assumes, because he saw or felt it as an innermost conviction, that the soul was the representative in the body of God in the universe. This is, of course, a position, not unscientific indeed, but extra-scientific, that is to say outside the sphere of science. If it is a true position, if, that is, the Soul is a *vera causa*, or real existence, scientific inquiry will not indeed reveal results incompatible with its existence—this it cannot do if science keeps to the truth of phenomena, which is always her aim—but the phenomena with which alone science has to do will not and cannot reveal the presence of a *vera causa* like the soul, for this is not a phenomenon, but an existence *per se*. Language of an ontological character, therefore, used by a man who, like Swedenborg, was essentially an ontologist, or a man who strove to reach the domain of things in themselves, such language may fairly enough be studied by the man of science, that is by him who, for his present purpose, confines himself entirely to phenomena. All that the scientific man has to do—it is a thing simple enough in theory, though difficult in practice sometimes—is to make and repeat the mental reservation where ontological phrases are used, that the ontological assumptions are a method of explaining the phenomena, which may or may not be correct, but that the subject-matter of science is only the phenomena. This kind of correction however is not peculiar to the kind of consideration we have instanced—to views that is concerning the soul and God. It is fundamental to all science and to all philosophy, and in point of fact it is the consideration which in principle lies at the bottom of the division of the domain of philosophy into two schools, the intuitional and the experimental. An instance or two will explain our meaning, before we pass to the scientific side of Swedenborg's views concerning the brain. In causation, science knows only succession, or perhaps invariable succession; but invariability itself can be predicated only experimentally, that is to say that it always has been so—but theoretically there may be an exception any day, though no one expects that there will be. *Necessary* connection, science never can predicate, because necessity is not in phenomena, but only succession, invariable within the limits of experience. The same considerations apply to the *power* assumed to lie behind phenomena of causation as Hume's billiard balls proved to all the world in Swedenborg's own day. The scientific reader who is at all acquainted with the history of philosophy will at once recall many other instances of terms used freely by science, which are calculated to raise similar questions. Matter, Force, Energy, are some of them, as well as those we have mentioned, and it is curious to observe that though, properly speaking, these are philosophical terms, it is Science which has been responsible for the very much wider currency which of late years they have obtained. Of course where Science does make use of them it ought fairly to be only as general names and for the purpose of avoiding unnecessary repetition, but it is full of instruc-

tion to analyse the very various uses that scientific men make of such terms and to trace out the large and even appalling amount of very loose thinking which they frequently cover. But (and this is most important) for the very reason that *veræ causæ* like God and the Soul can never appear as phenomena, for this very reason science can say nothing about them—least of all can she say they do not exist. If they do exist, she cannot see them, because her vision does not extend so far—they are out of her range.

The questions relating to their existence are questions not for her at all, but for ontology, or for philosophy; so true is it that the metaphysical questions are after and also beyond the physical, the phenomenal, the scientific. We do not say what weight should be attached to the fact that terms involving reference to ontological considerations are continually arising out of science. Nor do we attempt to raise any ontological discussion on them. Our present purpose is only to show what we therefore repeat, that such a *vera causa* as the soul, assuming its existence, can never appear in phenomena. But the language of him who assumes a *vera causa*, because by the eye of reasoning he sees it, will be intelligible to the scientific man who refuses to transcend the phenomena, provided he always keeps in mind the difference that exists between phenomena and modes of accounting for them by the assumption of independent existing *veræ causæ*. We may add that the learned editor of Swedenborg appears, though without explicitly stating it, to have kept this great distinction continually in view, there being few instances indeed in which it has been forgotten. On p. 740, however, in the course of his very elaborate note on the Chymical Laboratory of the Brain, he uses "animal spirit," viz. the fluid secreted by the brain as synonymous with "nervous force," placed indeed within inverted commas, a phrase suggestive rather of metaphysical than of physical considerations. Again, when on p. 660, Dr. Tafel says, "The mind by its influx into the brain is the cause of the motion of that organ," does he use the word *mind* in a scientific sense or a philosophical?

The following are some of the views held by Swedenborg on the Brain and its functions, and the statement of them will show both how frequently he anticipated modern discoveries, and also how many of his opinions are still more or less peculiar to him and his followers. If time and space permitted, it would be very instructive to inquire in how many of these views Swedenborg was correct, or how far at least the evidence appears to justify the conclusions to which he came. Some of this evidence will be referred to in this review, but it will be impossible to take up all the points, interesting and important though some of them undoubtedly are:—

1. Swedenborg taught that the motion of the brain is coincident with respiration, not with pulsation. The origination of this view is now universally (that is so far as it is held, for many physiologists of the present time do not seem to know of it)

attributed to J. Daniel Schlichting, a physician of Amsterdam, who wrote in 1750; but Swedenborg wrote between 1741 and 1744. We propose to devote some space to the discussion of this view of Swedenborg, because it is fundamental to his opinions, and because the most elaborate and learned note of Dr. Tafel enables us to do this in a most complete way. We will at present therefore only say that the complete statement of Swedenborg's position on the subject is that the dura mater undoubtedly pulsates with the movement of the heart, but that the underlying brain as undoubtedly moves synchronously with the respiration and not with the pulsation of the heart.

2. Swedenborg held that the respiratory motion of the brain and lungs extends to the extremities of the body. This discovery is generally attributed to Dr. Piégu, who wrote in 1846.

3. Although Swedenborg taught that the movement of the brain was synchronous with that of the respiration, he also held that it is independent of it. This point will be discussed in our general review.

4. Any student of Swedenborg will see that the term "animal spirits," so frequently used by old anatomists and old philosophical writers, is an expression not used only of a metaphysical idea analogous to *soul*, but of a veritable fluid, having an actual existence, and composed, according to Swedenborg in a very definite way, and fulfilling a very definite function or purpose in the economy. We confess to having had our conceptions much cleared in this particular by a perusal of the book.

5. Swedenborg taught that the anterior part of the cerebrum is the seat of the intellect. This is precisely the view adopted by modern science.

6. It is worthy of mention that Swedenborg's division of the brain is not quite that which obtains at the present time. His highest lobe is bounded by the marginal convolution of the quadrate lobule, his middle lobe by the lower part of the marginal convolution, and the gyrus fornicatus; and his third lobe is identical with the temporo-sphenoidal lobe. Keeping this in mind, we find him making this statement. The muscles "and actions which are in the ultimate parts of the body, and thus in the soles of the feet, depend more immediately upon the highest portions of the cerebrum; upon the middle lobe the muscles of the abdomen and thorax; and upon the third lobe those which belong to the face and head; for they seem to correspond to one another in an inverse ratio." The general correspondence of these statements with the positions of the most modern science cannot fail to strike the reader.

7. Swedenborg says, "The corpora striata are vicarious cerebra, and they succeed in the place of the cerebrum whenever it is deprived of its power of acting." And again: "The corpora striata initiate movements which at first originated with the cerebrum, and were voluntary; for it is a well-known fact that voluntary acts by daily habit become spontaneous, or that habit is like second nature." This language again is strikingly like that of modern science.

8. So also Swedenborg showed the connection of the corpora

quadrigemina with the sense of sight; and also what we may shortly call the commissural arrangement of the corpus callosum.

9. Key and Retzius have shown (1875–6) that the choroid plexuses of the lateral ventricles are made up of two leaves, of which the upper comes from the fimbria of the fornix and the lower one partly. Strange to say, Swedenborg said very much the same thing 135 years before.

10. Swedenborg says that in order that the brain may be relieved of the liquid which is perpetually expressed from its members and arterial vessels, the above liquid passes into the cavities of the nose through the openings in the cribriform plate of the ethmoid bone. This position also, though it is not accepted by medical men or by physiologists, appears to have been corroborated by experiments made by Key and Retzius.

On this we will only say, since space does not allow of our discussing the question fully, that there are cases coming from time to time under the notice of the medical man which appear to corroborate Swedenborg's statement. Patients frequently say (we have met such) that they are subject to a form of attack accompanied by pain in the frontal region, generally brought on by over-mental work and simulating a cold in the head. Such attacks pass off, and relief is at once experienced on the appearance of a clear discharge from the nostrils. Such persons have assured us that they were also subject to attacks of nasal catarrh, and that they could perfectly distinguish between the two, but that the attacks now spoken of were quite different, and that in their opinion the fluid comes from the inside of the head and not from the nasal mucous membrane.

11. Swedenborg held that the active parts of the brain are the cortical substance, and that "the brain is made up of as many similar forms and natures as it has discrete cortical parts." And further that "the cortical substance is so connected with the minute vessels of the brain, that you would believe that the cortical glands derive their origin from the vessels." Swedenborg did not say he had seen this structure, but, reasoning from analogy, he felt certain of his position that "the cortical glands hang down," and sprout forth from the sides and coatings of the producing and generating arteries, scarcely otherwise than as grapes and berries are wont to hang down," &c. Dr. Bevan Lewis, writing in 1877, says from his microscopic investigations, that a minute blood-vessel invariably runs in close contact with all the large nerve-cells; and that the peri-cellular sacs are laterally disposed along the sides of the smaller capillaries, in no case occupying a terminal position. This is a remarkable corroboration of the truth of Swedenborg's statements.

Lastly. Swedenborg's method of enquiry and the conclusions at which he arrived appeared to him to demand that some foramen or foramina must exist at the base of the fourth ventricle, through which the cerebro-spinal liquid is discharged from the ventricle; and they also seemed to him to require the presence of a channel from the fourth ventricle into the spinal cord. Since Swedenborg's time a foramen in the former position has been discovered, called

the foramen of Magendie, and also in the latter, where it is known as the central canal of the spinal cord.

As regards propositions 1, 2, and 3, on the Respiratory Motion of the Cerebrum, no mention whatever is made of this by such modern physiologists as Todd and Bowman, Carpenter, Michael Foster, Draper, Flint, Hermann or Ferrier. So that, speaking generally, we may say the view is not held by modern science. And yet Realdo Colombo (1559), Bartholin (1673), Wepfer (1681), Vieussens (1685), Baglivi (1701), Ridley (1703), Bellini (1714), Pacchioni (1721), and Fantoni (1738), all of them literary predecessors of Swedenborg, had held and taught this view.

The question thus arises: how did a view held so widely come to be no longer accepted? From the learned note of Dr. Tafel we learn the answer to this question. It was Pelletan, professor of physics in Paris, 1829, who, basing himself on the impossibility of the walls of the cranium lending themselves to the least extension, also on the state of repleteness in which the cranial cavity is at all times, and on the incompressibility of the liquids and the cerebral substance, laid down, *à priori*, this proposition, that *in the normal state with the adult these movements are an impossibility.* In short, Pelletan held and Bourgougnon (1839) thought he had proved experimentally that in the air-tight cranium, filled with incompressible fluids, and with brain substance which behaved as such, all motion was impossible. The so-called proof of the proposition was this. Bourgougnon trephined a hole in the cranium, and then screwed a ferrule to which a brass tube had been inserted, into the hole. In the interior of the tube there was a little metallic plate resting on the brain, which communicated the motion of the brain to a little lever with an elbow, answering for an indicator. This motion was seen to agree nicely with the beating of the heart, so long as the tube containing the lever was open. But as soon as it was filled with water (previously boiled, in order to exclude its air-particles) beyond a little tap fixed so as to intercept all communication with the atmosphere, on the tap being turned off, the movements of the little lever at once stopped. This gave rise to the conclusion that the motion of the brain exists only as long as there is an artificial communication with the atmosphere. Richet, writing in 1860, said he had repeated Bourgougnon's experiment, and found it correct; but argued that the inductions drawn from it were false, and that the experiment did not by any means prove what it was assumed to prove. He pointed to the condition of the dura mater in the vertebral cavity, and to the existence in its cervical portion of various venous plexuses which are backed by a compressible layer of a fatty liquid, in order to prove the capacity of the vertebral tube of expanding. But an experiment devised by Mosso (1877) seems to settle the question, and in the words of Dr. Vaillard, to have established the motion of the brain in the ossified cranium. He says, "Put your hand into a jar (through a lid made air-tight) furnished with two mouth-pieces. One of these mouth-pieces is closed with a very elastic membrane; the other supports a tube

with a tap, the tube being filled with water beyond the tap. It is easy to see that as long as the communication with the external air is kept up, the oscillations of the water consequent upon the increase and decrease in the volume of the hand are considerable in the tube where the tap is kept open, while there are scarcely any vibrations in the mouth-piece covered with an elastic membrane. Why is this? Evidently because the changes in the level are produced at that point where there is the least resistance, and because there is nothing to oppose itself to the manifestation of these changes in the tube which is in communication with the open air. On the other hand, the elasticity of the membrane of india-rubber covering the other opening, however weak it may seem, nevertheless opposes a serious obstacle to the movements of the column of water, all of which, in this case, are conveyed towards the open tube. This corresponds to the first part of Bourgougon's experiment, to that part where the movements of the brain are conspicuous. Let us now close the tap of the tube in which the oscillations had been so manifest. All motion now disappears, and it cannot be otherwise, since the column of water is not compressible. But has your hand on that account ceased to change in volume, and to swell each time when there is an afflux of blood? All you have to do is to examine the elastic membrane, which has hitherto remained motionless, and which now begins to heave and to subside by turns, in order to convince yourself of the fact that the oscillations still continue, and again manifest themselves where they meet with the feeblest resistance. The case is the same in the second part of Bourgougnon's experiment; if the lever which rests on the brain ceases to indicate any motion after the tap is closed, the reason is that the movements are conveyed towards those parts of the vertebral canal which are compressed."

The motion of the brain being thus established as a fact, the next consideration is of what sort is that motion. Up to the time of Swedenborg, although, as we have seen, there was no doubt about the motion (which was doubted only after his time) it was universally (?) held that that motion was coincident with pulsation. Swedenborg's views led him to the conclusion that it must be coincident with the respiration. He knew well enough that there was a motion of the brain coincident with pulsation also, but that motion he held was due to the dura mater, or rather to the outer layer of that membrane. The movement of the inner layer of the dura mater, on the other hand, corresponded with the motion proper of the brain and was coincident with the respiration. Now, in these particulars, it is very remarkable that later discovery has entirely justified Swedenborg's views. Some time ago the present writer abstracted for 'BRAIN' Professor Mosso's work on the motion of the brain, referring at length to the tracings obtained from that motion by the graphic method of Professor Marey. There is therefore the less reason to do here more than refer to his work, but it may be said that the labours of all the inquirers, by means of Professor Marey's graphic method—a method about which there

can be no mistake, since it is really self-registering, coincide in showing changes in the volume of the brain coincident with the respiration. There is no doubt about the pulsatile movement of the dura mater, but coincident with this there is also observed a heave and fall of the curve corresponding with the respiration. Dr. Tafel reproduces in his note tracings from Salathè (1876), François-Franck (1877), and Mosso (1881), which show this double motion beyond dispute. Nor is there any more inherent difficulty in understanding how such a double movement may take place, than in understanding, what every one knows, that the heavenly bodies have a movement round a centre such as the sun, at the same time that they revolve on their own axis.

Leaving this point, then, we pass on to say that Swedenborg held that the respiratory motion of the brain, although coincident with it, was not due to the respiration, but was inherent in the brain itself. Here also modern science confirms Swedenborg's position, for Professor Mosso shows that a change in the pulse of the brain (observed in the dura mater) does not depend by any means upon a change in the movements of respiration. "Thus" he says, "while I put Bertino through a process of multiplying 9 by 40, there was caused an increase in the elevation of his [animatory] cerebral pulse, while the increase in the motion of respiration ensued only after the increase in the volume of the brain had been accomplished." The conclusion of Swedenborg from this fact would be that the mind or the soul is the cause of the motion of the brain by its inflex into it. We shall not attempt to follow him into the regions of philosophy, which are foreign to our present purpose, but we apprehend that there is nothing unscientific in holding the view that the brain-cells have while living the property of self-movement, or, let us say, of motion. Cells in general, when examined under the microscope, even those which have no known or discoverable trace of nervous matter about them, may be seen to shrink and swell, to contract and dilate under the eye of the observer. If cells in general do this, science names the frequent repetition of the phenomenon as *mobility*, or, she sometimes says (it is just here that science is apt to get puzzled, and to use phenomenal expressions as if they applied to things in themselves), *cells have the property of contractility*. Now if in the scientific sense cells have the property of contractility, there is plainly nothing unscientific in Swedenborg's assumption that the respiratory motion of the brain is a property resident in the brain-cells fundamentally, and at least, as we have seen, Mosso's scientific inquiries are in keeping with this conclusion.[1]

[1] It was during the discussion of this very question, when the question was whether contraction and dilatation might be called the manifestation of the "Cell-soul," that Haeckel on a recent occasion indulged in that wild demonstration against Virchow, accusing his former master of breaking away from the teachings of his earlier days, and by his vehement and almost passionate language reminded us of nothing so much as a heart-broken and despairing lover undeservedly thrown over by the being he had adored. We believe that if Virchow, the scientific evolutionist, had been clearly distinguished from Virchow the philosopher, or spiritual teacher, all of this wild "scene" would have been avoided. But there

Arising out of Swedenborg's views on the self-originated respiratory motion of the brain naturally follows his position concerning the government of the cerebral arteries, which, according to him, are emancipated from the government of the heart, and have their blood-current moved in the stream of the animatory motion of the brain, which is synchronous with the respiration. There are three facts which corroborate the truth of Swedenborg's position, or, we may even say, prove it. First, the brain and cerebellum are supplied by the internal carotids and vertebrals, while the arteries of the dura mater come from the external carotids. Second, the internal carotids and vertebrals undergo a remarkable series of twistings, just before entering the brain, which can have the effect only of breaking the force of the cardiac blood-current, and emancipating the vessels from the heart's impulse. And, thirdly, they lose their muscular coats, and become dilated into various ampullæ which must have the same effect on the circulation contained within them. An examination of such modern writers as Quain, Carpenter and Key and Retzius tends strongly to confirm the truth of Swedenborg's views in this particular.

We propose to leave a consideration of Swedenborg's views as to the glandular structure of the brain for future discussion, as subsequent volumes of this important work appear. Meantime it must be remembered that the theory must be taken as a whole, the particular propositions which we have discussed concerning the motion of the brain being only parts of the whole, and such as, in the view of the author, could be no otherwise than he stated them, and as they were in fact, having regard to the part which the brain plays in the body.

A quotation from Swedenborg himself will fitly bring this review to a conclusion. It places his theory in a harmonious manner before the reader, and also states, what we have made only the most incidental reference to, his view as to the course taken by the animal spirit or fluid secreted by the brain acting in its glandular capacity.

"It is absurd to suppose that any action can begin in the middle of a fibre, and not in its first terminus. If then it begins in the first organs, it must inevitably begin in the cortical glands" (or nerve-cells) "for the fibres commence, and are conceived and produced in those glands, and the arterial vessels of the brain terminate also in them. Hence if the principles of motion exist in them, according to all physical and philosophical laws, as mutually confirmed by and confirming each other, those principles must necessarily commence by a kind of active, living, or locomotive, reciprocal force, that is, by a kind of expansion and constriction, or systole and diastole, such as we observe in a gross form in the lungs and heart; for the same conditions are involved, whether the spirit

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is more to be said than this, strong as it seems, in favour of Swedenborg's views as to the self-origination of the respiratory motion of the brain. Space fails indeed for the full consideration of the question, but we refer the reader who may feel interested in it to Dr. Tafel's note, pp. 670–677, and also to pp. 680–691, where he will find the whole subject historically and exhaustively treated.

is to be driven through the fibres, or the blood through the vessels. The blood cannot be driven through its arteries, without the reciprocal expansion and constriction of the heart; nor can the spirit be driven through the fibres, which are little canals and vessels analogous to the arteries, only more pure, without the reciprocal exhaustion and constriction of the cortical glands of the brain, which, on this account, deserve the appellation of pure corcula or little hearts. Assuming, or granting these points, the necessary consequence is, that every time the cortical and cineritious substance of the cerebrum, cerebellum, medulla oblongata, and spinal cord contracts, or constringes itself, the whole mass of these parts sinks down, and undergoes systole; but, on the other hand, undergoes diastole, when the same substance, I mean the whole congeries, expands. This is the animation of the brain—using the term brain in its widest acceptation—that corresponds to the respiration of the lungs. We must now proceed a step further. If the animal or nervous spirit, at the interval of the constriction of these organic substances—of the little hearts of the cerebrum—is expressed by the cerebrum through the nerves and nervous fibres, of course it is expressed by the cerebellum into its grand sympathetic nerves, the pneumo-gastric nerve, and the great sympathetic cords: and granting this, it follows that these nerves act during the same intervals upon the fibres of the pulmonary plexus, and upon the fibres of the costal nerves; which cannot fail on the instant to act upon their muscles and membranes; nor the latter to act upon the ribs, and thus upon the internal structure of the lungs. Hence it follows that the animation of the brain (using the term here again in its widest sense) must necesarily be coincident with the respiration of the lungs: and the fact is still more plainly declared by the influx of the fibres of the above mentioned cerebellar nerves, the pneumo-gastric, and the great sympathetic, into all the viscera of the abdomen; and by the motions of those viscera agreeing exactly, and keeping perfect time with the respiratory motions of the lungs."

No doubt there is here a mixture of scientific and metaphysical considerations, although the former largely preponderate; but we venture to say that the scientific reader who takes up the book will feel it well worth his while to separate the one from the other, as we have attempted to do, and that he will admit that the method of a writer who so long ago could make contributions to brain-physiology of so permanent a value, must have some qualities that entitle it to attention.

A. Rabagliati.

Critical Digests and Abstracts from Journals.

Bernhardt on Apoplectiform (and Epileptiform) Attacks in Tabes.—Bernhardt (*Archiv f. Psych.* Bd. xiv., p. 135) reports three cases of tabes in which peculiar apoplectiform attacks were noticed. The attacks came on suddenly, and lasted sometimes an hour, sometimes several days. In some instances there was loss of consciousness, in others consciousness was retained, and there was simply aphasia, or loss of power in one limb or in one side. Similar apoplectiform attacks are known to occur in alcoholism, senile dementia, disseminated sclerosis, and general paralysis, but the cases reported showed no symptoms of these diseases. The absence of any of the symptoms of general paralysis is particularly noteworthy, for it is well known that tabes and general paralysis are not unfrequently associated. It is possible, indeed, that in Bernhardt's cases symptoms of general paralysis would have ultimately shown themselves, as five or six years have been known to elapse before the symptoms of general paralysis supervened on those of tabes. But within the last few years Debove, Lecoq, and others of the French school have reported cases of apoplectiform and epileptiform attacks occurring in the course of pure cases of tabes; so that we must recognise these transient attacks of hemiplegia and aphasia as among the symptoms of tabes—often, indeed, as among its earliest symptoms. It is difficult to account for them; but this much is certain—that they do not betoken any serious brain lesion.

Westphal on a Disease resembling Cerebro-spinal Sclerosis.—Westphal (*Archiv f. Psych.* Bd. xiv., p. 87) reports with great fulness two cases which presented the symptoms of multiple cerebro-spinal sclerosis. There was paresis of the muscles of the extremities, the trunk, and the neck, with rigidity of the muscles, and as a result of this delayed and difficult movements; there were tremors when voluntary movements were made; there was "scanning speech" (in one case only, in the other it resembled rather the speech found in bulbar paralysis); the sensory disturbances

were slight and transient; the patellar reflex was evocable, and also the paradoxical contraction; and there were headache, vertigo, apoplectic and hemiplegic attacks, and mental symptoms, particularly dementia. The absence of nystagmus would be regarded by some as an important omission from a case of multiple cerebro-spinal sclerosis, but Westhal's experience is that it is frequently absent. In its place there was an abnormality in the movements of the ocular and facial muscles; the muscles responded slowly to the stimulus of volition, and the movements when commenced were slow in execution. This was well seen in the length of time it took to open and close the eyes. Both cases terminated fatally after a very chronic course. In one case a careful examination failed to reveal any cerebro-spinal lesion, in the other the cerebral pia mater was somewhat œdematous, the convolutions small, and the white matter of very firm consistence; but there was not any sclerosis, in the ordinary acceptation of the word. Westphal concludes from these two cases that there is a general neurosis (which, for want of a better name, he calls a pseudo-sclerosis) which cannot be distinguished, either in its symptoms or its course, from the disease known as multiple cerebro-spinal grey degeneration, or sclerosis.

Westhal adds some remarks on the so-called paradoxical contraction of a muscle brought about by its relaxation, the approximation of its points of origin and insertion. It is best seen when the foot is passively flexed; the foot remains flexed and adducted, owing to contraction of the tibialis anticus, and it may continue flexed for half an hour. In one of the cases reported in this paper, the paradoxical contraction was observed not only in the tibialis anticus, but in the flexors of the knee-joint, in the supinator longus of the forearm, in the muscles of the wrist and finger-joints. This condition is due to a change in the muscular tonus, and bears an analogy to Thomson's disease, but we as yet know very little about it.

Senator on Paralysis of the Fifth Nerve.—A man aged thirty-nine (*Archiv f. Psych.* Bd. xiii., p. 590) presented the following symptoms:—Complete anæsthesia of the left half of the face, mouth, and nose; loss of taste in the anterior part of the left side of the tongue; anæsthesia of the left cornea and conjunctiva, there being no blinking on touching the left eye, but if the surface of the right eye was touched the eyelids of both eyes closed, and the ordinary blinking movements of both eyes occurred as in health. Left conjunctiva much injected, cornea

softened and opaque, tension of the globe diminished. The only impairment of mobility was paresis of the left masseter. The diagnosis was lesion of the left trigeminus at some point between its exit from the pons and the gasserian ganglion. The patient gradually improved under the use of iodide of potassium, dry cupping, and blistering, but there appeared from time to time slight and transient swelling—first of the left knee-joint, then of the right knee-joint, and afterwards of both ankle-joints.

Senator regards the above as a case of neuro-paralytic keratitis, and makes some observations on this affection. Clinical facts oppose the theory of external irritation as the cause of the phenomenon, and the present case shows that the eyelids on the affected side still afford a considerable protection to the eye. Clinical facts equally oppose the idea that the arrested flow of the lachrymal secretion and the consequent drying of the cornea cause the ophthalmia. The only feasible explanation is that the appearances are due to some influences acting on the nutrition of the tissues, whether through the vaso-motor nerves or through actual trophic nerve fibres is not known.

Senator adduces this case in proof of the old view that the gustatory fibres of the chorda tympani take their origin in the trigeminus. The swelling of the joints he attributes to a vaso-motor or trophic disturbance; it certainly was not a rheumatic affection.

Moeli on the Reaction of the Pupil on Stimulation of the Sensory Nerves. It is well known that stimulation of the sensory nerves causes dilatation of the pupil. Moeli (*Archiv f. Psych.* Bd. xiii., p. 602) has almost never failed to obtain this result in women and children, though in some women over fifty the reaction was not so marked as in younger individuals. In men over fifty, ordinary stimulation failed to evoke it in a number of cases, but powerful electric stimuli generally succeeded. Moeli made observations on the condition of reaction in various nervous diseases. In *general paralysis* he found that where the pupils reacted well to the stimulus of light, sensory stimulation failed to produce dilatation of the pupil in 13 per cent. of the men; there was a decided dilatation in 74 per cent. men, and 100 per cent. women. Where the pupils did not react to light, there was no dilatation in 65 per cent. men, and 50 per cent. women; decided dilatation in 10 per cent. men, and 14 per cent. women. In cases of *tabes* with pupils not reacting to light, dilatation was obtained very rarely, and then it was very slight, and obtained only after strong stimulation. It is interesting to note that in

male general paralytics with retained patellar reflex the pupil did not react to light in 36 per cent., whereas in paralytics with absent patellar reflex the pupil did not react to light in as many as 83 per cent. The corresponding figures in women were 40 per cent. and 63 per cent. These figures suggest a connection between disease of the posterior column of the cord and the affection of the pupils. In *epilepsy*, the pupils dilated rapidly after even slight sensory stimulation. The dilatation of the pupil in these cases is not due, as Schiff and others have held, to "an actual cerebral sensation," for it is found during sleep, in coma, and in cases of hysteria with hemianæsthesia after stimulation of the anæsthetic side. And the absence of the pupil reaction must not, especially considering its frequency in tabes, be put down to disease of the cerebral cortex, as is too often done in cases of general paralysis.

Schwarz on the Skin and Tendon Reflexes.—In this paper (*Archiv f. Psych.* Bd. xiii., p. 621), Schwarz endeavours to support, on clinical grounds, the theory that the different spinal centres are related to different brain centres, and that the varying condition of the various spinal reflexes is due to the varying condition of the corresponding brain centres. He points out that where the brain centres are in a state of excitation, as in cases of cortical epilepsy with descending degeneration of the motor tracts (he reports several cases) there is increase of the tendon reflexes, especially directly after the motor discharge; the patellar reflex being generally increased on both sides, the ankle phenomenon being only observable on the side of the convulsions. The skin reflexes are increased, except after very severe convulsions, when they are diminished or absent. In cerebral hemiplegia the tendon reflexes are absent when there is simply paralysis, but heightened if the lesion has produced irritation of the motor strands. The best index of an irritative lesion of the motor strands is the heightening of the tendon reflexes; it often precedes the appearance of contracture by several days. The skin reflexes are always absent or much diminished.

Schwarz distinguishes between the achilles-tendon reflex and the ankle-clonus. He relates a case in which passive dorsal flexion of the foot produced ankle-clonus merely, while percussion of the achilles-tendon produced, in addition to plantar flexion of the foot, general muscular spasms of both legs. The paper concludes with an examination of the various theories that have been offered in explanation of the phenomenon of the tendon-reflex.

De Jonge on the Tumour of the Medulla Oblongata with Diabetes Mellitus.—In this case (*Archiv f. Psych.* Bd. xiii., p. 658) a tumour the size of a small bean, and consisting of a tubercle caseated in the centre, projected from the left side of the medulla oblongata; it reached from just below the olivary body to the first cervical nerve, and destroyed the substantia gelatinosa of the posterior cornu between these two limits. All other parts of the medulla and cord were intact. The case was one of tuberculosis of the lung and mesenteric glands, in which diabetes was a subsequent and secondary symptom, and the author argues that the tubercle in the medulla oblongata was the cause of the diabetes. He cites eleven cases of diabetes with lesion of the medulla oblongata and neighbouring parts, in all of which the lesion was situated considerably higher than the tubercle in question, and he concludes that the tubercle did not affect the so-called diabetes centre itself, but probably implicated the nerve strands in their course from it to the periphery.

Kirchhoff on Hemiathetosis.—Kirchhoff (*Archiv. f. Psych.* Bd. xiii., p. 582) reports a case of hemiathetosis of the left side appearing suddenly in a patient of intemperate habits, suffering from cardiac dilatation and cirrhosis of the liver with ascites and œdema. The case differed from ordinary cases of athetosis in that the movements were not confined to the hand and foot, but were participated in by the eyes, jaw, and face. After a time slight athetoid movements appeared in the right hand. There were no sensory symptoms, and only transient and inconsiderable paresis of the left hand. The patient died three weeks after the appearance of the hemiathetosis, and a careful *post-mortem* examination failed to show any coarse brain lesion or any miliary apoplexies or embolisms. Delicate false membranes were found in the arachnoid sac of each side, but these Kirchhoff refuses to consider as the cause of the unilateral athetosis.

Flatten on Diabetes Insipidus.—Flatten (*Archiv f. Psych.* Bd. xiii., p. 671) records a case of diabetes insipidus associated with paralysis of the external rectus of the left eye and paresis of the same muscle of the right eye. The diagnosis was a focus of softening a little below the nucleus of the left abducens nerve, destroying the root of the nerve and extending across the middle line towards the right abducens. Gayet has reported a case of diabetes insipidus with paralysis of the right abducens, and Kaemnitz a case of diabetes mellitus with paralysis of the same nerve.

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THE CEREBELLAR CORTEX.[1]

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Dr. Gaule, under whom I have worked, and whom I have much to thank for his kind assistance, advised me to use Weigert's säure fuchsin method (*Centrbl. für die med. Wiss.* 1882, p. 753), which has produced excellent results. The first sections were made from pieces of cerebellum, one centim. square, hardened in three per cent. solution of bichromate of potash and kept in a hot chamber at 35° C. for 4–8 days. They were then washed in water half an hour, then placed in alcohol 24 hours, and then for 24–28 hours in a concentrated aqueous solution of säure fuchsin, also at a temperature of 35° C. The pieces were then slightly washed in water and put into strong alcohol and dehydrated, imbedded in paraffin and cut into sections, which were then fixed to object slides, the paraffin dissolved out,[2] and the sections washed out according to Weigert's method with water and alkaline alcohol.

In the second series of preparations they were further stained with nigrosine after the above treatment; and these preparations proved the best. The third series were hardened in the same way, but after fixing the sections on the slides, they were stained by hæmatoxylin and eocene. This latter method of double staining has already been employed by Denissenko. In the fourth method employed, the pieces of cerebellum were hardened by $\frac{1}{4}$ % chromic acid, imbedded unstained, and the sections fixed on the object slide. The specimens were taken from the cerebellum of man (adult and new-born) dog, rat, rabbit, hen and pigeon. My description is chiefly concerned with the dog, in which all the elements are most clearly shown. The human cerebellar cortex is the most developed, and as it is much richer in medullated fibres, and the medullated sheaths are better developed, it would undoubtedly give better preparations, but unfortunately it is impossible to harden the pieces directly after death and so obviate post-mortem changes. And great stress is laid on the fact that in the preparations used for this paper, in less than an hour after death, the pieces were in the hardening fluid in the warm chamber, which was used to introduce a quicker diffusion in the substance of the piece.

[1] This article is an extract from a paper published in the 'Archiv für Anatomie und Physiologie' (Physiologische Abtheilung) and from work done in Professor Ludwig's Physiological Institute in Leipzig.

[2] Canini, in these Archive, 1883, p. 155.

1.—Granule Layer.

Basis of description, säure fuchsin and nigrosine preparations. In these the medullated fibres are seen, as described in Fig. 1 and 2 *a*, as a black or grey thread enclosed in the medullary sheath stained red. It is very important to note that the axis cylinder is dark and the medullated sheath red. If the säure fuchsin be not sufficiently washed out, other fibres which are not medullated are dyed red, but these have not the dark, central fibre which can be stained by nigrosine or hæmatoxylin, and my description refers only to these fibres.

Fig. 1 is a longitudinal section of a lobule of the cerebellar cortex seen under a low power. Along the centre of this run the medullated fibres, radiating out and passing through the granule layer in the direction of the Purkinje cells. These fibres have been described by Deiters,[1] who maintained the connection of the axis cylinder process of these cells with a medullated fibre. Koschewnikoff[2] has isolated Purkinje's cells in connection with a medullated fibre. Hadlich[3] says that from the white medullated-fibre centre, fibres proceed to the grey red granule layer, divide, and pass as a principal nerve process into the large ganglion cell, into every cell one fibre. Golgi[4] makes the axis cylinder process, in its course through the granule layer, give off branches at right angles. Other descriptions are of a similar nature. But how very indistinctly the fibres have been seen is shown by the very contradictory statements, as for instance some say: that the axis-cylinder runs direct to the medullary centre; that it runs in a horizontal direction; that this axis-cylinder soon gets a medullated sheath, or that this occurs at a great distance from the cell; and that the fibre is fine,—or thick. Nearly all maintain that it divides. Hadlich, and, later, Denissenko make reservations on account of possible deceptions. Hadlich's statements seem to me the most correct.

Fig. 1 shows a clear picture of the whole arrangement of the nerve-fibres; in each lobule some of them can be traced uninterruptedly in their whole course to the immediate vicinity of the Purkinje cells. The fibres leave the medullary centre with the slightest change of direction. At the summit of the lobule they radiate out like the outstretched fingers of the hand. At the lateral part of the lobule they ascend slantingly in a slight curve through the granule layer. They run at definite distances from each other, corresponding to the distances between the Purkinje cells, to which their number, as far as can be ascertained, corresponds. At the bottom of the sulci the fibres are closely aggregated and several pass through the granule layer together.

[1] Deiters, 'Untersuchungen über Gehirn und Rückenmark.' Herausgegebene v. M. Schultze, 1868.

[2] Koschewnikoff, "Axencylinderfortsatz der Nervenzellen im Kleinhirn des Kalbes;" M. Schultze's 'Archiv,' 1869, Bd. v.

[3] Hadlich, "Mittheilung über den Bau der menschlichen Kleinhirnrinde." 'Arch. für mikr. Anat.,' 1869, Bd. vi.

[4] Golgi, 'Centralbl. für die med. Wiss.' 1879.

These fibres make a bend at the border of the molecular layer, running parallel with it for a short distance (see fig. 1); they do not cross each other and do not branch; but keeping the same thickness, they run straight through the granule layer with some slight wavings in their course. Their axis-cylinder is thick, the medullated sheath slightly varicose, often presenting a jointed appearance. In short, these fibres have in the granule layer the same formation they had in the medullary centre. Each remains an isolated medullated fibre which, unbranched and independently, directs its way towards a particular ganglion cell. One can hardly doubt that these are their nerve fibres. Sections are not very adapted to trace the passage of a fibre into a structure of so much greater size. If the sections are very thin, the chances of finding the right spot are extremely small, and this difficulty is increased as the fibres always change their direction before entering the cell. Isolated preparations show more easily the connection, but then it is not certain that the fibres are of the same nature. I have therefore not been satisfied till I could confirm also in section preparations the connection of the Purkinje cells with one of these fibres.

It can therefore now be said for certain that every Purkinje ganglion cell is in connection with a distinct, isolated, medullated nerve fibre. Besides the above described fibres, there are others, which have already been seen and which caused the different opinions concerning the fibres going to the ganglion cells, as it was not known that there were two different kinds of fibres. Only Hadlich [1] has as yet expressed this idea, and laid stress on the fact that the many divisions described must refer to fibres of the second kind. The second form of fibre produces a plexus which traverses the granule layer in all directions, passing into the medullary centre on one side, and into the molecular layer on the other. These fibres branch about and anastomose, they are of different thickness, from the finest calibre up to that of the first kind of fibres, their medullated sheath is less developed, is always varicose, often interrupted, and sometimes the fuchsine reaction entirely fails. In man these fibres appear far more numerous and better developed than in the dog, when stained by säure fuchsin. Perhaps age makes some difference.

It is quite clear that these fibres differ from those of the first kind. The latter pass through them without being connected with them. Probably all branched fibres belong to the same species and have the same function. I shall designate the direct fibres of the first kind, joining ganglion cells as *straight*, or *unbranched* fibres, those of the second kind as *branched* or *anastomosing* fibres.

The spaces between the fibres in the granule layer are filled up by the closely packed cells. Formerly these cells were all called granules, and their cellular nature was doubted. Denissenko has shown that we have here two kinds of cells, one stained by hæmatoxylin, the other by eosine. The hæmatoxylin cells correspond

[1] Hadlich, op. cit. p. 20.

to those formerly called granules, but they possess a nucleus and protoplasm, and therefore the attributes of a cell. Denissenko regards these as connective tissue cells, whereas the eocene cells are connected with nerve-fibres.[1] The reason why this formation was not recognised before, was because the cells are so tightly packed. Very thin sections have to be stained by a dye which will colour the protoplasm, for this is always present in the form of a very small layer around a relatively large nucleus. The protoplasm has numerous processes, very fine or thicker threads, which form a thick network with the threads of other cells.

The threads refract light strongly and have a sharp contour, the meshes are fine. They are best seen, where the cells lie less packed together, at the borders of the granule-layer. Towards the medullary centre there is no sharply-defined boundary, and as in the granule layer there are everywhere fibres, so in the medullary centre there are similar cells scattered everywhere. But here the protoplasm is more abundant, the threads thicker, and the meshes of the network are very much larger and join with the narrow network inside the granule layer, resembling the fine framework in the white substance of the spinal cord. The nucleus of these cells is very large and characteristic. It possesses a distinct thread-like appearance, with the above-mentioned method of hardening. The threads lie grouped in all kinds of forms in a round clear space. If these nuclei were richer in colouring matter, and if characteristic stages could be found, the idea of nucleus-division might be entertained. Some of the nuclei are certainly larger and their contour more distinct. The mass which is coloured by hæmatoxylin is naturally that of the nucleus threads, while the protoplasm, even when coloured by nigrosine, only appears as a narrow line running round the nuclear spot. Between the cells the medullated fibres make their way, and their medullated sheath comes into most intimate contact with them. The cell threads often seem to penetrate between the separate particles of the medullated sheath. Denissenko describes these cells as grouped round openings from which channels proceed, whose walls are likewise lined by these cells. This appearance is seen when the cells lie particularly close together. These channels are only the medullated fibres which have penetrated between the cells, whilst the place where the fibres cross each other gives rise to the apparent openings. If the cells are less closely arranged, the spaces occupied by these fibres are less compressed, and this presents the appearance of cells in groups surrounded by fibres.

In these spaces other structures are seen, which colour with eocene and nigrosine, and called by Denissenko, *eocene cells*. They seem to be interposed in the course of the medullated fibres, which cross over the cells. They are often really interposed, as can be seen by using nigrosine and säure fuchsin, when the cells become the same colour as the axis cylinder which is then seen

[1] Henle and Merkel have already compared the granules to the cells of reticulated connective tissue, and have therefore described them correctly according to their appearance.

to be in connection with them, while the medullated sheath is interrupted.

Their cellular character is doubtful, as I can find no nucleus in most of them. The larger ones resemble multipolar ganglion cells by their form and size, as also by their connection with nerve fibres; and as it is often difficult to discover a nucleus in ganglion cells when the protoplasm is coloured, these are probably ganglion cells. The smaller of these cells are often deficient in any cellular character and appear to be thickenings along the axis cylinder.

I think I can with certainty explain the use of the hæmatoxylin cells, viz. that of glia-cells. These have not only nuclei, but are cells with protoplasm and processes; the processes form a network which extends through the whole granule layer, and which is also connected with a similar network in the medullary centre.

These appearances are just the criterion of glia-cells, and if one accepts glia-cells at all, they must be cells like those above described. Schwalbe has lately objected to the glia-cells generally, from the hypothesis that the cells can be separated from the fibres of the finest network. But these objections are mostly theoretical. Besides, in my preparations, the network of the fibres proceeds from the body of the cell (see plate Fig. 2, *b*) and this agrees with what Henle and Merkel give in their work on the supporting structure of the central nervous system. Also in the description of the supporting framework of the molecular layer I have only to confirm their observations, agreeing with them that these cells are glia-cells, but doubting that they are cells of reticulative connective tissue, or that they may become ganglion cells. Since the embryologists have shown that the central nervous system proceeds from the ectoderm, and that there exists a thorough separation between the epithelial and connective tissue structures, the "archiblastic and parablastic" formations, it is probable from embryological researches, and what we know of its chemical nature, that the glia is an epithelial formation.

The glia stands in relation to the ganglion cells and their protoplasmic processes, as the medullated sheath does to the axis cylinder of the peripheral fibres. It is a continuation of this latter, and there are forms of transition. The cellular elements are common to both; from this can be explained, what might be considered an objection to the definition of these granule cells as glia-cells, viz., their accumulation into distinct regular layers. Where numerous fibres lose their medullated sheath (those of the first type), or acquire it (those of the second type,) there are to be found accumulated the cellular elements which are common to the glia and the medullated sheath, and where this occurs in regular planes in the cerebellum, the position of the cells is found to correspond.

2.—Molecular Layer.

a. The ground Substance.—This layer owes its name to the peculiar substance forming its foundation. The older authors

describe it as a finely granulated mass, in which a finer structure is not recognisable; however Fromman has already shown it to be a sponge substance built out of a network of threads. Kühne and Ewald have proved that we have here a formation of a peculiar chemical structure, analogous to the horny framework of the medullated sheath of the peripheral nerves. Schwalbe describes the substance in his 'Lehrbuch der Neurologie' as a fine network, the fine trabeculæ being composed of neuro-keratine. This opinion is also borne out by my plates, Fig. 2, *b–d*. The clear substance lying in the enclosed spaces of this network becomes, with the means used to clear up the medullated substance, as clear as this latter; the osmic-acid method cannot be applied, as the distinction between the trabeculæ and its meshes only comes into view with very thin sections, and these can only be made after treatment with alcohol. The säure fuchsin method, however, produces a very characteristic action on erythrophile substances. In many of the mesh-spaces, but not in all, a fine red line bounds the clear contents, in the same way as seen in the cross-section of nerve-fibres.

These red-bounded spaces are larger or smaller in size, and sometimes several in a row. But they are not sections of fibres, as can be seen from the absence of any axis cylinder; neither are there, where they lie, so many fibres visible, which is easily proved by altering the direction of the section. It thus becomes probable that as this network corresponds to the horny framework of the medullated sheath, so also this intermediate substance corresponds to the myelin; but not chemically, for even the medulla substance of the periphery differs from that of the central nervous system in not re-acting to säure fuchsin; and we must consider that the relation of the neuroglia of the central nervous system differs more from the peripheral nerves than does the medulla of the fibres of the central nervous system. There are, however, many transitions, viz., the above described varicose sheaths of the branched fibres, which often form separate round formations and the glia-nets containing erythrophile substance. Another analogy becomes visible, when we consider the cellular elements of both, viz., the glia and the medullated sheath. Ranvier has acquainted us with the cells of the medullated sheath, and he concludes that the myelin is enclosed in the meshes of their protoplasm. Towards the sheath of Schwann and the axis-cylinder the protoplasm is thickened to form the inner and outer protoplasmic sheath. When this theory was proposed, Kühne and Ewald had not yet discovered their horny sheath, which probably corresponds to Ranvier's protoplasmic sheath, if neuro-keratine be substituted for protoplasm. I think it probable that from the protoplasm of the young cell, neuro-keratine is formed by differentiation, as is similarly described by Waldeyer in the horny change of the epidermis cells. There, a stroma of keratine threads forms itself out of the protoplasm of the cell, while in its meshes, a fatty substance, eleidin, is deposited. It is difficult to distinguish between the sheath and the framework. Either can be resolved into neuro-keratine. Probably transitions

between the two will be found. I have also tried to convince myself in preparations of the horny sheath, treated by Kühne's method and coloured by hæmatoxylin and nigrosine, whether the neuro-keratine threads really are in connection with the nuclei of the medullated sheaths, and I can assert that one can really see this, so far as it can be observed. Assuming the horny framework of the peripheral nervous system to be a formation of cells, its relation to that of the central nervous system appears all the more assured. For not only the network of the granule layer already described, but also the network of the molecular layer, appears as glia-cells. The connection between both is best observed in the collection of glia-cells to be found at the base of the molecular layer. When from any reason, the molecular and granule layers are separated—which may be caused even by pressure of the cover-glass—there remains one or more layers of cells separated with the molecular layer. The separation occurs below the Purkinje cells, where the unbranched fibres bend round and spread out. Here, the layer of glia-cells attached to the molecular layer forms a membrane, by joining their processes, to support both the fibres and blood-vessels spread out under it, and the Purkinje cells resting on it, and to form also the groundwork of the molecular substance. In Fig. 2 *b*, this is shown in section. Its cells are generally larger than those of the granule layer, as are also their protoplasm and processes, which here become very distinct. Many are pyramidal or pear-shaped, with their broader basis turned to the granule layer, and form, with the long processes given off from the glia-nets of the granule layer, that membrane of the molecular layer which may be represented as a flat network broken up by the cells interspersed in its meshes. Through this membrane pass all the numerous formations on their way from the granule to the molecular layer, and vice versâ. As the name of *limitans externa* has been adopted by Henle and Merkel for the layer of the glia-nets which is in contact with the pia mater, and this layer, by its formation and origin being completely analogous to the above-mentioned membrane on the inner surface of the molecular layer, I propose to call the latter *limitans interna*. Some of the processes from its cells, which are vertical to the limitans interna and go off from the apex of the cells, are particularly thick and can be traced far into the glia-network; and at regular intervals these processes are still thicker and reach to the limitans externa, to which they are attached by a flattened pedicle and serve as supporting pillars to the molecular layer. These were discovered by Bergmann and described by Henle and Merkel. They have also described the limitans externa as a special membrane, separable from the pia mater, and consisting of glia threads. Obersteiner has described one or two cell layers under the pia mater in the new-born animal, from which layers is developed the limitans externa, as well as the supporting pillars; probably the limitans externa has the same character in the new-born as the limitans interna in the adult, so that the pedicles are remains of pyramidal cells, whose apex processes formed supporting pillars

and basal processes the network of the limitans externa. The network between the membrane is also probably the remains of the cells described by Obersteiner, as filling up the embryonal molecular layer, and which have been absorbed with the growth of the network; as the network is too extended to have been formed from the supporting fibres, or from the glia-cells scattered in the molecular layer. Perhaps the protoplasm of the embryonal cells may have been converted into neuro-keratine. Two further points about the glia-cells remain to be discussed. (1) Their relation to the ganglion cells and their processes; (2) their relation to the connective tissue.

(1) We have already mentioned that the glia-cells are accumulated round the ganglion cells (see Fig. 2 *b*). Whilst the ganglion cells at their lower part rest on the limitans interna, they are packed at the sides by these heaped up glia-cells, so that the cells, together with the limiting membrane, closely surround a hollow in which the ganglion cell lies. At the upper and lower part the processes pass out. This walling-in is seen best when the Purkinje cell has been detached from its space, which is then seen spun over with a network of fine fibres, which are continued at the sides into the bodies of the glia-cells. This capsule is thus formed by a net of neuro-keratine threads, supported by a denser accumulation of cells similar to that found in the limitans interna. A somewhat similar capsule has been described by Denissenko and by former authors. Of course such a capsule consisting of a network will allow the cell a free connection on all sides; there is, however, a special condition for the axis cylinder and the protoplasmic processes. The former seems to acquire a continuation of this capsule as a sheath, which is connected with the medullated sheath of the nerve-fibre. But I have no definite drawings on this point. As is evident from Fig. 2, *b*, *c*, everywhere in the course of the protoplasmic processes the glia-nets surrounding them become thickened, so that numerous threads seem to be attached to them, whilst on each side, the meshes of the glia-nets become larger. This attachment of glia-threads has been described by Hadlich and other authors, who state that the protoplasmic processes appear rough, or that extremely fine threads go off from them laterally.

This attachment is however only apparent; it is in reality only a tube-like thickening of the network around these processes, forming a sheath of neuro-keratine threads, continuous with the capsule of the ganglion cells. This appearance resembles the axis-cylinder of the peripheral nerve fibre (when treated according to Kühne's method), which is closely surrounded by a thickening of the horny structure, decribed by Kühne as the inner horny sheath, and here the keratine threads often seem to join the axis-cylinder itself. The broad meshes of the glia-nets on the sides of the protoplasm processes correspond to the space between the inner and outer horny sheaths, which is also traversed by a few threads. A formation analogous to the outer horny sheath is absent in the glia. The resemblance between the relation of the protoplasmic process to its glia, and the relation of the axis-cylinder to its horny

sheath helps very much the comprehension of the central nervous system. But the sheaths in the glia are not isolated, they are in connection everywhere, and are only interrupted by the branching out of the processes. In the glia, the horny component part is unduly in excess of the thread network, as compared with the fatty part enclosed in the meshes: and the fatty substance is not identical with myelin of the peripheral nerves. As Weigert's reaction is only sometimes shown, probably different gradations of myeline exist in the nerve-fibres of the periphery and in those of the central nervous system, where varicose fibres occur, increasing as they become finer.

(2) It is not easy to establish the boundary between the glia and the connective tissue. When they were still unconditionally classed together, it was observed that there by no means existed an immediate connection between the pia and the glia lying under it. Henle and Merkel—who in their treatise assume that glia, nerve elements and connective tissue are produced out of the same materials—describe, however, that the glia limits itself by a lamina limitans externa, and that this is easily detached from the pia, being separated by spaces traversed by single threads,—subarachnoid lymph spaces. A retraction of the glia from the connective tissue sheaths of the vessels leads to the production of the peri-vascular spaces. The glia therefore is only in connection at single points with the connective tissue, which is necessary to give support to the whole framework. In short there seem to be connecting-fibres only between the pia and the limitans externa, as these fibres do not sink deeper into the glia-network.

The connective tissue of the pia may be compared with the Schwann sheath of the peripheral nerves, if the glia be compared with the medullated sheath; and as between the Schwann primitive sheath, and the medullated sheath, so between the connective tissue and the glia, there exists only a loose connection; from this it does not follow that the glia might not histologically be reckoned with connective tissue. But this depends entirely on whether or no the glia-cells are descended from epithelium cells of the ectoderm, for our present classification of the tissue is essentially generic. From the above, it is no longer possible to treat of the glia as an undefined boundary between nervous and connective tissue. We have then in the nervous system three distinct systems lying close together:

1. Axis cylinders.	Ganglion cells.	Protoplasm processes.
2. Medullated sheaths.	Glia-cells.	Glia net-works.
3. Schwann's sheath.	Pia mater.	Connective tissue-sheath.

Of these systems the first only can be designated as nervous.

b. The Nervous elements.—One link in the chain of this nervous system fails us. The branched processes of the Purkinje cells must enter again into connection with nerve fibres. It is easy to trace the branches to close to the limitans externa, but here every direct trace is lost. Rindfleisch and Stricker suppose them to become lost in the ground-substance of the neuroglia, but

this has been very much contested, as it is against the physiological demand for a definite course. In my preparations I find no indication of a connection of the ganglion-cell processes with the threads of the glia network. A second view makes the processes end in peculiar nuclei, which lie at the periphery of the molecular layer. I have found it difficult to discover what form of cells Denissenko means by these peripheral nuclei, for at the limitans externa or towards the inner part of the periphery, there are very few cells to be found in the adult animal. Numerous cells are present in the young animal only. Denissenko calls these cells nuclei, and yet states that they are very little coloured by nuclei staining fluids, and further, that in some animals, fibres spring from them which penetrate vertically the molecular layer. The latter fibres correspond to the supporting fibres of the glia, and I consider that the so-called peripheral nuclei are the pedicles with which the supporting fibres attach themselves to the limitans externa. The degree of coloration mentioned by Denissenko agrees also with these pedicles, which might also be called nuclei, as they are possibly the remains of cells; but with the processes of the Purkinje cells these pedicles are not connected.

This view of Denissenko agrees with Golgi's, who makes the processes of the ganglion cells end in connective tissue corpuscles at the periphery, which Denissenko even conjectures to be partly identical with his peripheral nuclei. With the exception of the sheaths of the vessels, I must decidedly dispute the presence of real connective tissue corpuscles at the periphery of the molecular substance on the inner side of the limitans externa. A third view makes the processes of the periphery bend round and run back to the granule layer; and to prove this, certain fine straight fibres, which traverse the molecular layer vertically to the periphery, have been pointed out as these processes (Boll); but the greater number of these fibres can be traced to the limitans externa where they end in pedicles. They are the supporting fibres, as others have already shown.

Lastly, Hadlich has suggested that the ganglion cell processes return again in the same form. This appears at first difficult to contradict, as the processes may be either running out or returning. But Hadlich has already conjectured that there might be two kinds of nerve-fibres, one entering into connection with the axis cylinder of Purkinje's cells, the other with the already mentioned returning branched processes. The latter therefore would not return to ganglion cells, but join directly with nerve fibres. In this way we have a distinct criterion by which to recognise the out-going from the in-going fibres, and a proof of whether they really exist. I have looked through my preparations and found that there are numerous places where, in the deeper parts of the molecular layer, all ganglion cell processes can be traced to their corresponding Purkinje cells. This precludes the possibility that the protoplasmic processes, after spreading themselves out at the periphery, should collect together and return in the same form to the granule layer, to join with nerve-fibres. Another hypothesis assumes that the returning processes

reassemble in the ganglion cells, of which there are two kinds, and which are not to be distinguished in their outward appearance. But this is contradicted by the fact that the branched medullated fibres can be traced far into the molecular layer. The object of these fibres could not be seen, if the protoplasmic processes return to a ganglion cell lying at the border of the granule layer before joining the nerve fibres. Besides this the number of unbranched fibres is probably just as large as that of the ganglion cells, so that we can drop the hypothesis of two kinds of ganglion cells. If the number of the unbranched fibres is just as many as the Purkinje cells, and if they end at the granule layer, whilst the branched fibres extend into the molecular layer, the probability becomes very great that this cell is in connection with both kinds of fibres, with those in the granule layer by its axis cylinder process, with the others in the molecular by its branched protoplasmic processes.

Fig. 3 is drawn from a section cut parallel to the surface, and therefore at right angles to the direction of sections hitherto shown. The drawing represents the bottom of a sulcus, so that not only the molecular layer, but also the Purkinje cells and a portion of the granule layer are visible on each side. The medullated fibres appear on the flat surface of the section, running in two directions at right angles to each other, and round the Purkinje cells are numerous fibres passing into the molecular layer. The section here given is very thin, or else the fibres would be very numerous, as is the case in thick sections, where it is difficult to see individual fibres. This horizontal plexus is most dense in the deeper parts of the molecular layer, around and immediately above the Purkinje cells. But also in the more superficial planes, fibres running in the same direction can be seen, and the better the reaction has succeeded, the further does their region extend. They are all much finer than the unbranched fibres, but of the same calibre as the finer branched fibres. In the corresponding vertical sections of the lobule, fibres are seen in bundles close round the capsule of the Purkinje cells and passing into the molecular layer. Little of the above described horizontal plexus can be seen in these vertical sections, as the one set of fibres is cut across (compare Fig. 2, *b*), while the other runs parallel to the surface. On the other hand there are fibres visible, which descend vertically in the third plane. These are also fine and often run close to the supporting fibres. Generally these cannot be traced further than through half the molecular layer. Beyond that, although erythrophile substance is met with, it is not arranged in the form of fibre sheaths, but in isolated rings, without any connection. Probably these single rings must stand in some relation to the continuation of the system of medullated fibres.

As the connection of the fibres of the molecular layer with the fibres of the second kind in the granule layer is very distinct, we may say that there exists here a fibre system, which, starting as a plexus from the medullary centre, branches through the granule layer, until the finest fibres are lost in the molecular layer, whilst their medullated substance can be traced further than the axis-cylinder.

As regards the connection of the separate elements it is easy to establish the following points of a scheme:

1. *Each unbranched fibre is connected with each Purkinje cell. The axis-cylinder passes into the protoplasm of the cell, the medullated sheath into the glia-like capsule of the cell.*

2. *The axis-cylinder becomes converted into a number of fibrils in the cell, which pass into the branched protoplasmic process. The fibrils run in the process, which is surrounded by a glia-sheath, as completely distinct threads as far as the periphery. In the branching of this process, the numerous fibrils, contained in it where it leaves the cell, are gradually distributed until they become isolated.*

My preparations show that the arrangement of the ganglion cells corresponds to M. Schultze's scheme. Obersteiner has already described this for the Purkinje cells. Sometimes fibrils cross each other at the points where the processes branch.

3. *The fibrils thus isolated bend round at an angle of* 90° (*not* 180°), *spread themselves out in a plane lying parallel to the surface, and re-arranging themselves in definite order as fibres surrounded by a medullated sheath, run, with frequent interchange between the fibres, in the form of a plexus back to the medullary centre.*

This third point of the scheme contains, of course, much that is hypothetical. But there can be little doubt that the fibres of the second system serve to establish somehow a connection with the branched processes of the ganglion cells. To explain why the fibres should not bend at an angle of 180°, instead of 90°, I must refer to Obersteiner's discovery that the protoplasmic processes of the Purkinje cells only spread out in a plane at right angles to the surface, and also to the direction of the lobule and the plane of the medullated fibre layer. On the other hand, the medullated fibres of the molecular layer run in planes parallel to the surface, and at right angles to the planes of the processes. The processes only need now to bend round at an angle of 90° to pass into the plane of the fibres. Certainly other fibres are required which shall turn at an angle of 90° and connect the higher lying planes with the lower, but these fibres would be coarser and medullated. The strictly angular arrangement of the elements of the cerebellum is remarkable, both in the nervous elements and in the supporting structure. The important point is that we do not see the connecting elements between the ends of the protoplasmic processes and the beginning of the nerve fibres; and in the absence of any really decisive result, some other solution of the problem is possible. Perhaps future research will reveal that just at that point where the continuity of the elements is lost, an arrangement intervenes which we cannot now recognise.

Further, there are medullated fibres under the pia mater lying on the limitans externa, broad fibres of a similar character to those of the medullary centre.

The same fibres may be seen passing through the molecular layer in a slanting direction. A second collection of medullated fibres of the same character lies half-way up the molecular substance. Both run parallel to the medullary centre. As these fibres are

Fig. 1.

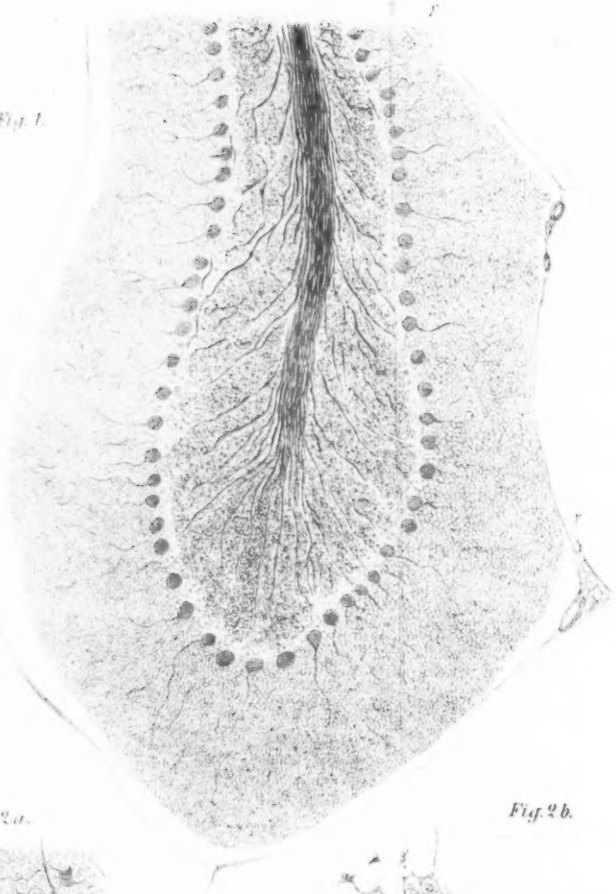


Fig. 2b.

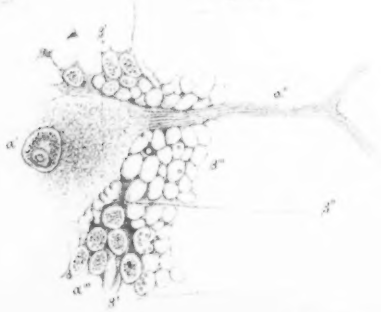


Fig. 2a.

Fig. 2b¹.

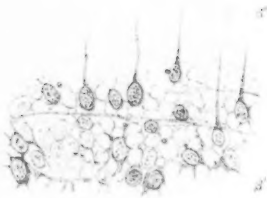


Fig. 2c.

Fig. 3.

Fig. 2b².

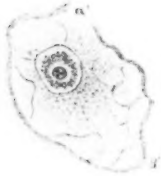


Fig. 2c.

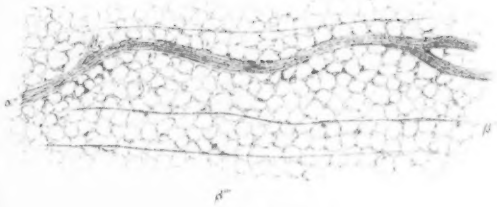
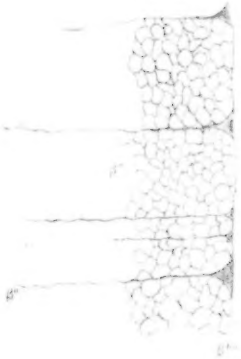


Fig. 2d.



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only found in a small minority of the lobules, they are probably an aberration of the medullary centre.

Sometimes the Purkinje cells of the same lobule differ in appearance. Some show, as seen in Fig. 2 *b*, a distinct nucleus, with the nucleolar contents surrounded by a ring of nuclear substance, and in the protoplasm, a distinct fibrillous formation which completely fills up the glia-capsule and in the double-staining with nigrosine and säure fuchsin are stained a light grey colour by the former only.

In the other kind a nucleus is hardly to be distinguished, and no fibril-formation; the cell appears as a homogeneous body which remains dyed by säure fuchsin, even after washing out by alkaline alcohol. The cells and their processes are slightly retracted from the glia, and the relation between the capsule and its sheath is not quite so distinct. It is not probable that there are here two kinds of ganglion cells, as the difference in the cells is only seen in some of the lobules. It is hardly due either to different action of the hardening re-agent or to the fact of one cell dying before the other, as an external influence would act differently on the cells of two distant lobules, but not on those of the same lobule. We must conclude that the condition of cells must have originally been different at the moment of death.

EXPLANATION OF THE PLATES.

α Unbranched broad nerve-fibres of the first kind.
α' Purkinje's cells.
α'' Their protoplasmic processes.
α''' Branched fibres of the second kind.
β Glia-cells of the granule layer (granules, glia-cells).
β' Cells of the membrana limitans interna.
β'' Supporting fibres (long radiating processes of the glia-cells).
β''' Glia network (short branched processes of the glia-cells).
β'''' Membrana limitans externa.
γ Pia Mater.
δ Non-nucleated structures of the granule layer, stained by Eocene and Nigrosin (Eocene-cells).

GENERAL VIEW MAGNIFIED WITH A LOW POWER.

FIG. 1. Longitudinal section of a lobule from the cerebellar cortex. Medullary centre—internal or granule layer—external or molecular layer. The pia mater with its blood-vessels is slightly separated. Shows the radiation of the unbranched fibres from the medullary-centre through the granule layer to the Purkinje's cells.

FIG. 2. Details of the same section seen by a higher power ($\frac{1}{12}$ oil immersion) showing:

a. Granule layer.
b. Boundary between granule and molecular layers.
c. Middle of the molecular layer.
d. Outer part of the molecular layer.

The figs. 2 *a–d* are to be taken in series.

2. *a*, shows fibres of the first kind, α, and of the second kind α''' between the glia-cells, β, and the eocene cells, δ.

2 *b*. A Purkinje's cell, α' with its protoplasmic process, α'' which breaks up into fibrils. The glia-cells of the lamina interna, β', some of them large and rich in protoplasm, are heaped around the Purkinje's cell. The glia network β''' proceeds from the processes of the glia-cells, some supporting fibres β'' are here not seen joining the cells. In the meshes of the glia-nets are seen sections of medullated fibres of the second kind, α''', stained red.

2 b^1. The glia-cells, in the interval between two Purkinje's cells, form by their intimate connection the limitans interna, β'. Some pyramidal-shaped glia-cells are elongated into processes which extend direct to the periphery and form supporting fibres β''.

2 b^2. A Purkinje's cell in the glia capsule. The cell is seen, so as to show clearly its capsule. The threads of the glia are partly visible coursing over the cell.

2 *c*. Glia network in the middle of the molecular layer. Very clear broad trabeculæ of the network, β''', which is traversed by parallel supporting-fibres β'', and also by a branching process from a Purkinje's cell, showing fibrils, α''.

2 c^1. A supporting fibre, β'', in connection with one of the solitary glia-cells found in this region.

2 *d*. The periphery of the lobule, with the limitans externa β''' cut across at right angles, and with the supporting fibres, β'', attached to it by broad pedicles; between them is the glia network.

Fig. 3. Horizontal section (parallel to the surface) through a lobule of the cerebellar cortex near the boundary between the molecular and granule layers, which is depicted at the periphery of the drawing, so that the Purkinje's cells appear surrounded by medullated fibres of the second kind which having reached the molecular layer, spread out in the plane of the section, in two directions at right angles to each other.

These plates are faithful representations of the preparations, and are not in any way schematic.